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REVIEW

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Physiology of the Heart and Circulation and Its Clinical Application in Physical Medicine

Part II of a symposium presented at the 27th annual conference of the American Physical Therapy Association in Cleveland, Ohio, June 26-30, 1950

Physical Medicine in Diseases of Peripheral Circulation — WISE

The Effect of Temperature and Various Methods Used in Physical Medicine to Increase Temperature on Local Circulation — HINES and RANDALL

Physical Medicine in the Clinical Aspects of Local Heat Application — BOUMAN

The Role of Circulation in the Physiology of Heat Regulation — KELLER

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To Our Readers

and Best Wishes for the New Year

from

The Physical Therapy Review

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Volume 30 **December 1950** **Number 12**

Physiology Symposium, Part II*

Physical Medicine in Diseases of Peripheral Circulation

Charles S. Wise, M.D.

No one can question the value of physical modalities in the treatment of peripheral vascular disease. The multiplicity of physical measures employed in this group of diseases, however, suggests that no single physical modality for any condition affecting the peripheral circulation is curative, in itself. Physical therapy, therefore, must be integrated in the over-all care of the patient, and frequently is an adjunct to other medical and surgical practices. The more we understand about the physiology and responses of the peripheral circulation, to physical and pharmacological stimulae of various types, the more rational will be our approach to the handling of these patients. The advances made during the past decade in treating peripheral vascular diseases have been due largely to two general trends. First, there has been an increasing interest in improving our physiological tests and measurements, giving us a more objective evaluation of the results of treatment, and second, the concepts and principles of rehabilitation tend to focus our thinking on the patient as a whole, rather than on the treatment of a single disease process.

How can the physical therapist contribute most effectively to the management of the patient with peripheral vascular disease? I should like to center this discussion on three principal subjects, with which all of us in the field of physical medicine should become better acquainted, and which, I am sure, will add to the results obtained in the management of this group of patients.

First, a basic knowledge of the various types of peripheral vascular disease processes, the pathological changes, as well as the physiological disturbances; second, a knowledge of the various physical modalities which are available, including their technics of administration, as well as their limitations; third, a broad viewpoint concerning the rehabilitation of the patient with peripheral vascular disorders—all are essential to the proper handling of this group of patients.

Before classifying and describing some of the most frequently encountered abnormalities of the

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peripheral vascular system, I should like to stress the importance of the early detection and diagnosis of peripheral vascular disease. The ability of the physiatrist, and, in some instances, the physical therapist, to detect undiagnosed vascular disease is the first line of defense against mismanagement in these conditions. Many undiagnosed cases are referred to the physical therapy department for the treatment of suspected musculoskeletal, or sometimes neurological, disturbances. Muscular cramps, for example, secondary to organic vascular insufficiency, are treated far differently than muscle spasm or muscle pain due to other causes. Failure to detect these differences early in the course of a patient's illness may result in harm rather than benefit. There is also a group of vasospastic diseases which have their basis in emotional conflicts, and long courses of physical therapy may perpetuate rather than relieve the underlying condition.

With this in mind, then, let us classify briefly the various types of peripheral vascular disorders.

We can divide these disorders anatomically, into two large groups—first, those affecting the arteries and arterioles, and second, those primarily concerned with the venous side of the circulation.

The arterial disorders, in general, fall into two categories—functional disorders and organic disturbances. Functional disorders include those diseases which are primarily due to increased vasospasm or vasodilatation; they are, fundamentally, physiological abnormalities in the degree of vascular tone present. In this group, however, despite the presence of a primarily physiological abnormality, these disturbances may be followed by structural changes, due, in most instances, to blood flow deficiencies over an extended period of time. On the other hand, the organic group of disorders, although primarily they involve structural changes in the arteries and arterioles, are frequently, if not always, associated with some functional vasospasm or vasodilatation, which may contribute to the pathological picture. Thus, although for purposes of classification, we divide the disorder into these two large groups, there is considerable overlapping on both sides.

Probably the most widely studied, if not the most frequent of the functional disorders, are those grouped under the classification of *Raynaud's syndrome*. The characteristic vasospasm in this disorder is generally initiated either by exposure of the digits to cold, or by emotional stress. Severe local pain in the fingers, associated with marked blanching, is almost pathognomonic of this disease state. Although this phenomenon was originally described as a disease, it has been encountered in a wide variety of clinical conditions and is now considered, in the great majority of patients, as a syndrome, or phenomenon, associated with more than one underlying etiology. The exact mechanism of this Raynaud's phenomenon has been disputed. Some authors feel it is due largely to abnormalities in the autonomic nervous system; whereas other workers feel that it is a local disorder in the smaller arteries of the digits, making them unusually sensitive to either cold, emotional stress, or circulating adrenalin. The mildest type of Raynaud's syndrome, or something very closely approximating it, is seen in the so-called hereditary cold hands. This is a type of vasomotor reaction occurring in many normal persons on exposure to cold, and it usually disappears quickly with warmth.

Very rarely, if ever, are trophic skin changes observed in this group of patients. In another type of patient, the syndrome or phenomenon may be precipitated by his vocation, particularly if it involves the use of vibrating hand tools. A group of patients with *pneumatic hammer disease* (in which the use of small vibrating tools precipitated attacks of Raynaud's phenomenon), whose symptoms usually disappeared when this type of work was given up, has been noted. In more severe instances of Raynaud's phenomenon, trophic changes —such as shiny appearance of the skin, deformity of the fingernails, superficial infection, and later, gangrene—may occur. These trophic changes have been classified as a *sclerodactylia*, and they resemble, pathologically, the changes found in *scleroderma*, which as we all know may involve the other portions of the body, as well as the digits.

Treatment of this group of disorders, of course, varies with the severity of the disease and the stage in which it is encountered. The avoidance of exposure to cold, or even slight chilling, the avoidance of emotional stress, and the use of local heat in early cases, may be sufficient to cause vasodilatation. In many instances, however, where simple measures are not adequate to control repeated attacks, and where early trophic changes are suspected, preganglionic sympathectomy has been employed. Most workers agree that, despite preganglionic sympathectomy, progression may still take place. This is in accord with the predominant view: that Raynaud's phenomenon is primarily a local disorder of the vascular system, rather than primarily one of the autonomic nerve supply.

Two other functional vascular disorders should be mentioned in passing—*acrocyanosis* and *livedo*

reticularis. As the name implies, the former consists of mottling peripheral cyanosis, which is induced or accentuated by cold, and affects the hands and feet symmetrically, with very few or no symptoms or serious consequences. It is usually associated with increased sweating of the palms of the hands. These patients usually require very little in the way of treatment—reassurance that serious circulatory disturbance is not present, and that avoidance of cold or emotional stress may be all that is necessary.

Livedo reticularis is a vasospastic disorder characterized by a cyanotic blotching of the skin, most frequently on the feet and calves, and again is not a serious disorder; only rarely is it associated with any trophic disturbances of the skin. The cause is unknown and as far as treatment is concerned, very little should be offered because of the mildness of the disorder itself.

Erythromelalgia is the last functional disorder to be mentioned. It is extremely rare, characterized by attacks of bilateral burning pain in the feet or hands, associated with marked redness of the skin depending upon the degree of arteriolar dilatation present. It is usually precipitated by those stimulae which normally induce physiological vasodilatation—such as local heat, exercise, or prolonged standing. It is similar in appearance, and possibly in underlying mechanism, to the reactive hyperemia which takes place after exposure to cold. The treatment again is avoidance of the precipitating factors mentioned above. The use of local cool applications and elevation of the extremity during the attack may be prescribed, but, in general, no specific therapy has been evolved.

The second large group of disorders of arteries and arterioles is grouped under the organic peripheral vascular diseases. *Thromboangiitis obliterans*, commonly referred to as Buerger's disease, is a serious progressive disorder involving the medium and small arteries and arterioles in a rather characteristic, pathological pattern. The disease occurs primarily in young individuals, between the ages of twenty and forty, and is confined primarily to males. The cause is unknown. The disease may start with a migrating phlebitis, that is, inflammation of the veins, but the diagnosis is usually made when the arterial system is involved. It begins primarily as an inflammation of the walls of the arteries, gradually involving the lumen of the vessel and secondary thrombosis. The chronic inflammation may involve the artery, vein, and accompanying nerves. In the early stages of this disease there is considerable vasospasm present; coldness of the extremity, pallor, and even intermittent claudication may be prominent. Depending upon the degree of collateral circulation which can be established, varying trophic changes occur, and as we have all seen, gangrene and loss of part or an entire extremity may be the end result.

A second organic arterial disease, probably the most frequent condition encountered in this group, is that of *arteriosclerotic peripheral vascular disease*. This condition is generally the cause of circulatory disturbance in patients above the age of fifty. In contrast to thromboangiitis obliterans, it affects both male and female. Pathologically, there are degenerative changes in the wall of the artery or arteriole, with atheromatous plaques leading to closure of the lumen, and thrombosis with eventual organization of the clot and obstruction of circulation. Calcification may occur and be seen in the X ray. It is important to note, however, that even in the face of marked changes in the larger and medium-sized arteries, as visualized by X ray, or palpated, clinically adequate collateral circulation and adequate blood flow in the smaller vessels may prevent trophic changes or disabling symptoms. Thus we cannot always correlate the severity of the involvement of the larger blood vessels with the amount of disability or trophic skin changes to be expected. In diabetics, we all know that there is an increased incidence of arteriosclerotic and degenerative vascular changes; these usually occur in an earlier age group, thus suggesting a metabolic factor involved in the arteriosclerotic disorder.

Immersion foot is an organic arterial disorder studied most carefully during the recent war. The diagnosis is obvious, occurring after prolonged exposure to cold and dampness. Methods for management of the acute immersion foot have improved during the past decade, when cleanliness, elevation, and slow warming of the extremity, avoidance of local heat, and the use of anticoagulants have definitely improved the over-all prognosis. In the chronic stage, the treatment by means of physical methods is the same as for any organic arteriolar insufficiency. Frostbite, on the other hand, is a clinical disorder, following exposure to intense cold, associated with actual freezing of the skin and subcutaneous tissues.

Other organic arterial diseases will be mentioned simply in passing. These are *arterial embolism*, *thrombosis*, *arterial venous aneurism* and *anastomosis*, and *glomus tumors*.

Diseases which affect the venous portion of the peripheral circulation are largely organic in nature.

Primarily we deal with varicose veins and thrombophlebitis. In the former there is anatomic alteration of the valves of the veins, and dilatation of the walls, secondary to the increased hydrostatic pressure. In thrombophlebitis there is an inflammatory disturbance of the walls of the veins, followed by thrombosis. We must distinguish thrombophlebitis, where there is an inflammatory process, from the condition called phlebothrombosis, where no inflammation is observed in the wall of the vein but thrombae occur, probably secondary to stagnation of blood in the larger veins, particularly in the pelvis and lower extremities of the bedridden patient. In the latter instance these thrombae are loosely attached and the danger of embolism is greater. Early postoperative ambulation, and the use of bed exercises in the convalescent or chronically ill patient, have resulted in a significant lowering of the incidence of thrombophlebitis in this group of patients. The adequate and early use of anticoagulant therapy has also diminished the hazards of a fatal embolus which may be the result of a detached thrombus in either of the above mentioned conditions.

With this very brief description of the principal diseases affecting the peripheral circulation, let us describe a rational approach in the management of these patients by physical medical methods. We have three primary objectives in the management of these patients—first, the prevention of tissue necrosis by alleviating the disproportion between the demands of tissue metabolism and the diminished blood supply, second, maintaining viability in tissues not already irreversibly damaged, and third, rehabilitating the patient as a whole.

In most of the arterial diseases mentioned above, blood flow to the extremity is diminished by varying degrees of vasoconstriction. In the organic diseases, however, there are irreparable structural changes in the arteries. Needless to say, any factors which cause increased vasoconstriction—such as cold, even slight chilling, emotional stress, or prolonged standing—must be avoided in this group of patients. On the other hand, any physical modality which may increase the local metabolic demands of tissue beyond the capacity of the diseased peripheral circulation should be avoided. In the latter case, irreparable damage can be done to the tissues and necrosis and gangrene may result. With this in mind, I believe that all forms of local heat, whether one uses diathermy, luminous heat, or conduction heating, are definitely contraindicated in the presence of organic arterial insufficiency. Other forms of reflex vasodilatation should be employed.

The danger of applying heat locally to a relatively ischemic extremity cannot be stressed too vigorously, particularly to nurses and attendants of patients. The physical therapist, when employing heat to the elderly patient for other reasons, should bear in mind the dangers inherent in peripheral circulatory disease. We still see too many patients with local areas of gangrene and trophic changes in the skin, resulting from the ill-advised use of heat on a relatively innocent lesion. One must be particularly cautious in treating a minor infection in the feet of an elderly or diabetic patient. The patient should be advised to avoid local use of hot, wet soaks, or electrically heated pads, which would result in extension of infection and precipitate necrosis of the tissue because of inadequate circulation and the increased metabolic demands as a result of local thermal therapy. In most instances we rely primarily on the avoidance of chilling and conserving normal body and skin temperature by having the patient wear warm socks and clothing. Where increased vasodilatation is desired, reflex vasodilatation can be obtained by local heat, applied in any one of several methods, to either the trunk, abdomen, or uninvolved extremity. Even the thermostatically controlled heat-cradle, in my opinion, is not a safe method for inducing vasodilatation in a patient with organic vascular insufficiency.

The objective in treating the patient with peripheral vascular disease is, of course, the prevention of tissue destruction. It seems to me that proper orientation of the patient, in details of foot hygiene, proper shoe-fitting, avoidance of chilling, and use of warm socks, and simple methods of reflex vasodilatation are probably more effective in the great majority of patients than any mechanical apparatus which, of necessity, can be used only over limited periods of time, and frequently at great expense to the patient.

Active, non-weight-bearing exercises should be stressed. Bed exercises, either of the Buerger-Allen type or simple muscle-setting exercises, hip-knee flexion, ankle curling, toe flexion and extension (particularly for the patient who is only semi-ambulatory)—all are excellent methods for maintaining optimum blood flow to the involved extremities without any inherent dangers.

In my experience, the *whirlpool bath*, when available for periods of twenty to thirty minutes once or twice daily, can be employed safely in organic arterial disease where the temperature is carefully controlled not to exceed 97° F. The mechanical stimulation of the agitated water acts

as a mild superficial massage, increasing local vasodilatation. Where ulcerative lesions are present, the mechanical washing away of superficial infected material may aid in the healing of these ulcerated areas. In the conservative regimen for the care of the patient with arterial insufficiency, the absolute avoidance of smoking should be emphasized without qualification.

Although these simple, conservative measures described above do not appear as impressive or dramatic as some of the mechanical apparatus which have been devised and are being used in the treatment of peripheral vascular disease, the success or failure of treatment, regardless of the modality employed, will depend largely upon how well the patient cooperates in this type of regimen. No single form of therapy can succeed where the good conservative regimen cannot be established. The use of various mechanical procedures to improve development of collateral circulation and improve local blood flow have received conflicting reports in the literature, but should be mentioned.

The *pavex boot*, or passive vascular exercise, involves the principle of exposing the affected limb to alternate positive and negative pressure. There is no unanimity to the rationale for the use of this apparatus. Some experimental evidence certainly indicates that there may be an increase in the size or number of useful collateral arteries developed as a result of treatment. Clinical reports are contradictory, however. Probably the most gratifying results are obtained in those patients suffering from a sudden occlusion of a large artery to an extremity. It appears that in these instances the proper use of a pavex boot can relieve the marked reflex vasoconstriction which follows an acute embolus. However, where any phlebitis is present, or in the presence of any infection, it is felt that this method is contraindicated. It does not appear to me to be rational, however, that in a patient with chronic arterial disease any significant change can be expected by the means of a mechanical procedure, applied for only a brief period of thirty minutes to an hour, once daily, or even less frequently. What the patient does during the other twenty-three hours each day is more important. I believe some of the variations in the results reported may depend largely on the technic employed by various workers. In almost no instance, however, has the pavex boot been used exclusively, without any other adjunct therapy. Thus it is difficult to evaluate the results. We have not used this type of treatment in our clinic. We feel that equally good results can be obtained with less expense and difficulty for the patient.

In more recent years the pavex boot has been superseded by *intermittent application of venous occluding pressure*. A simple, intermittent, venous-occlusion apparatus is available. Some workers believe that the mechanism involved is a filling and stretching of the vascular tree as a result of the compression of the venous circulation, with the release of vasodilating substances when pressure is removed. However, there is not very convincing evidence to back this assertion. In spite of lack of good physiological evidence that circulation is improved with this method, reports continue to appear claiming clinical improvement following its use. We have occasionally used this method on patients with intermittent claudication and impending gangrene but rarely do we feel that improvement in a patient's condition can be attributed to this type of therapy alone.

Mecholyl and histamine iontophoresis are still effective means of increasing vasodilatation, particularly in the superficial tissues of the skin. In refractory ulcers, due to either organic or functional vascular insufficiency these vasodilators are effective in accelerating healing. Recently, however, the increasing use of priscoline hydrochloride, intravenously, has enabled one to obtain equally satisfactory results with perhaps less inconvenience to the patient. In certain patients, who develop undesirable side effects from this medication, however, mecholyl and histamine iontophoresis are still used. *Contrast baths* have been advocated in the past, as a means of obtaining increased blood flow, but this cumbersome technic is in no way superior and may be less effective than the simple conservative methods described above.

The Sanders oscillating bed is an expensive mechanical apparatus which alternately tilts the patient from the head-down to feet-down positions, without expenditure of effort on the part of the patient. This seems to be even more effective than the Buerger's or postural exercises and when available can be used to advantage for patients with impending gangrene which fails to respond to other methods.

In general, the interest of most physiatrists today is turning from emphasis on mechanical apparatus, as a cure or prophylaxis in the management of the peripheral-vascular-disease patient, and turning more toward a total rehabilitation of the patient by emphasizing the simpler, conservative measures of treatment. As mentioned previously, emotional stress and strain, and worry over the patient's future may have a very deleterious effect

by producing increased and prolonged vasoconstriction. We feel, then, that it is important to reorient the patient, to relieve his apprehension, and to assure him that under a good, conservative regimen the probability of serious sequellae is made less likely.

The treatment of peripheral vascular disease has become increasingly efficient because of the cooperation between the internist, the surgeon, and the physiatrist. Diseases are recognized earlier and treatment begun before irreversible damage leads to loss of an extremity. The fundamental framework of treatment rests on a good physiological basis. In judging a method of treatment, we should be guided by objective evidence of its efficiency, its physiological soundness, and its simplicity, by the permanence of response. It is because of these general principles, rather than any specific method, that the prognosis in the patient with peripheral vascular disease is better today than a decade ago.

The Effect of Temperature and Various Methods Used in Physical Medicine to Increase Temperature on Local Circulation

Harry M. Hines, Ph.D., and Barbara Feucht Randall, M.S.

There is an axiom in physiology which states that adjustments are being made in the body continually, for the purpose of providing an adequate blood flow either for the organism as a whole or for any of its parts. Physiologic mechanisms exist which serve to regulate the volume of blood flow to tissues in accordance with their ever-changing metabolic and physical requirements.

An increase in the volume of blood flow through an organ or tissue will tend to provide the structure in question with additional quantities of food, oxygen, and heat and to remove excess quantities of metabolic wastes, including carbon dioxide and heat. An augmented blood flow may serve several purposes. It will lessen or delay the onset of fatigue and exhaustion, and thereby provide for greater functional performance and endurance. Through an increased supply of oxygen, it will tend to lessen or prevent the occurrence of anoxic pain and to reduce the accumulation of excess fluid or edema. An improvement in the circulation may serve to arrest or prevent functional impairment and structural damage which might otherwise take place in tissues as a consequence of an inadequate blood supply. Examples of the latter include gangrene, ulcers, and fibrosis, which may occur in peripheral vascular diseases, anuria from inadequate renal circulation, and myocardial degeneration, cardiac arrythmia, and fibrillation from inadequate coronary circulation.

Various physiologic mechanisms exist in order to provide an increased blood flow to either widespread or local areas in the body. The former is best exemplified by such exercises as running or brisk walking, in which the increased blood flow through active muscles is made possible by an increased cardiac output and by a redistribution of blood from relatively inactive to active areas. A rise of body temperature is usually associated with an increased blood flow through the cutaneous vessels. Local infection is associated with an active hyperemia in the contiguous tissues. The above represent only a few of the many instances where circulatory adjustments are made for the functional benefit of tissues.

A number of therapeutic measures may be employed for augmenting the blood flow through body tissues. These measures include active exercise; passive manipulation brought about by either stretching, massage, intermittent occlusion, or posture changes; denervation of blood vessels; vasodilator drugs; and temperature. Active exercise is

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a most effective measure for increasing the blood flow through skeletal muscles. However, it must be pointed out that exercise also increases the oxygen requirement of muscle by amounts which exceed those made available by augmented transport, thereby increasing, rather than alleviating, tissue anoxia. Sympathectomy is usually followed by an increase in blood flow through the denervated structure due to the release of the blood vessels from vasoconstrictor control. However, this effect on blood flow may be transient, due to the fact that the smooth muscle of denervated vessels soon develops supersensitivity to circulating hormones such as adrenalin, which in some tissues may reduce or nullify the increase in blood flow which follows denervation. Furthermore, the denervated structure is handicapped with respect to its temperature regulation because of loss of control over blood vessel caliber and sweat secretion. Many peripheral-acting drugs are effective for causing vasodilatation and increased blood flow through certain areas. As a rule, such drugs cause effective vasodilatation only when given in doses sufficient to cause undesirable circulatory side-reactions. For these reasons drugs are not the therapy of choice when local vasodilatation is desired.

Various modalities of heat have long been employed for increasing blood flow, and many of the alleged beneficial effects of heat treatments have been attributed to effects on blood flow. It is recognized that the methods which are available for measuring blood flow in the human are quite inadequate. For instance, the plethysmographic methods in general use are incapable of revealing whether an increase in blood flow is due to dilatation of blood vessels in the superficial or in the deep tissues, or in both areas. Much of our information concerning the effect of heat on blood flow has been obtained from the experiments on laboratory animals, under conditions which are not desirable for studies on human subjects. Information from such sources should be transferred to the human subject with caution and reservation.

Many claims have been made for the relative merits of the various modalities for increasing the temperature of tissues. These modalities include artificial fever, near and far infra-red, contact heating, diathermy, microwaves, and supersonics. It is generally recognized that the effects of heat are qualitatively the same regardless of their mode of production or transfer. Infra-red and contact heating are very effective for elevating the temperature of superficial tissues, but some degree of heating in the deeper tissues may result from conduction of heat from the warmer superficial tissues. However, some form of electromagnetic wave energy is usually preferred when appreciable amounts of deep tissue heating are desired. The longer the electromagnetic wave length, the deeper will be its penetration. Heating of living tissues by supersonic energy has not as yet reached the state of development which will permit its use in the treatment of human subjects with safety.

Temperature changes may affect blood flow through tissues through the operation of one or more of several mechanisms. Stimulation of the end-organs of warmth activate depressor nerve fibers which increase the activity of vasodilator centers, and concomitantly inhibit the activity of vasoconstrictor centers, thus permitting either a local or systemic dilatation of blood vessels. On the other hand, the activation of cold receptors and pain acceptors usually elicits either general or local vasoconstriction and decreased blood flow, through increased activity of the vasoconstrictor and inhibition of the dilator mechanisms. The effect of heat treatments on blood flow will, through these mechanisms, be determined by the algebraic sum of the effects on antagonistically acting peripheral receptors, and will be abolished or prevented in an area following sympathectomy. Heat through direct action may relax the smooth muscle of blood vessels and permit increased blood flow. A rise in the temperature of tissues increases their metabolic activity, and accordingly their rate of oxygen consumption and carbon dioxide production. Anoxia and metabolites increase blood flow by direct action; they produce relaxation of the smooth muscles of blood vessels. This means that, if heat therapy is to improve the effective blood flow through tissues, it must produce sufficient increases in flow to satisfy the circulatory requirements of an augmented metabolism. Otherwise it might intensify, rather than reduce, tissue anoxia.

The quantitative effect of heat therapy on blood flow will depend upon a number of factors which will be briefly discussed. The type of tissue to be treated is important, inasmuch as different tissues vary as to their vascular patterns and their mode of control. Thus the blood flow through the skeletal muscles is regulated in accordance with the metabolic requirements under different levels of activity; whereas blood flow through the skin serves to meet the metabolic requirements of that tissue, and, in addition, serves as a means of regulating heat loss.

The state of blood-vessel tone existing at the time of treatment has a bearing on the response to heat. If the blood vessels are near maximal dilatation, the effect of heat in eliciting relaxa-

tion will be less than when a state of marked vasoconstriction pre-exists. Evidence from animal experimentation points to the necessity of a certain critical degree of tissue-temperature elevation, which is necessary before significant increases in blood flow occur. These studies suggest that the explanation for the alleged therapeutic benefits from light doses of diathermy must be sought in effects other than circulatory, inasmuch as much heavier doses are required to produce significant increases in blood flow. An increase in the temperature of the circulating blood will elicit a dilatation of the cutaneous blood vessels over widespread areas. This effect of heat should be considered quite apart from the effect of heat on blood flow in the tissues contiguous to its application. Although the over-all effect of exercise is to increase the blood flow through active muscles, it is known that the presence of a strong tetanic contraction may be associated with either a partial or complete ischemia, because of impedance to flow offered by the contracted skeletal muscle fibers upon the walls of blood vessels. Thus the increase in blood flow occurs either between the phases of rhythmical contraction or after their cessation. The blood flow through skeletal muscles in a state of prolonged shortening or spasticity is significantly less than that in normal muscle. This means that, if the application of heat brings about a change in the state of tonus and extent of muscle shortening, it will influence blood flow because of mechanical conditions incident to changes in intramuscular tension.

Local heat therapy may be of such a degree as to result in the activation of the receptors of both pressor and depressor sensory fibers. The effect of such treatments on blood flow through a given structure would be the algebraic sum of these two antagonistic actions and might cause either an increase or a decrease in blood flow. It may be postulated that, during treatments with infra-red, certain pain acceptors, located in the hot spots of irradiated areas, may become trigger areas which operate at temperatures slightly below those eliciting disagreeable pain and cause reflex vasoconstriction. The sense of pain, although a valuable mechanism for warning against impending damage to tissues from overheating, may not be relied upon as a guide for the optimum tissue temperature level necessary for affecting blood flow.

It appears that several methods are at our disposal for elevating the temperature of tissues. These modalities are reasonably satisfactory and safe if certain precautions are observed. Uncertainty exists as to the quantity and quality of the effects of therapeutic heat upon blood flow. Further investigations are necessary before a decision can be made as to the underlying mechanisms involved in the therapeutic responses to heat. Evidence exists which suggests that the effects of therapeutic heat upon tissue metabolism, pain stimulus and acceptors, and upon muscle tonus may be more important in some instances than the circulatory changes which so far have been demonstrated.

Physical Medicine in the Clinical Aspects of Local Heat Application

Harry D. Bouman, M.D.

The application of heat in clinical physical medicine has in many instances become a more or less routine procedure, and it is well to reconsider our procedures in the light of our modern knowledge of physiology of circulation. In the previous paper, Dr. Hines has shown us the problems of local heat regulation in physiology and has also pointed out some of the difficulties in an intelligent application of heat in its different forms. I think he has pointed a way to the solution of a number of problems which are still before us. I want to review for you some of the accepted clinical procedures, in the light of the physiology of local circulation as it has been presented to us by Dr. Hines. Obviously much of what I have to say is familiar to all of you, but I hope that it will refresh your memory and stimulate discussion during the question period afterwards.

I think it is good to realize from the start that there is an essential difference between the local application of heat and the application of general heat. In the local application, body temperatures are not raised significantly, and, therefore, influences on the temperature regulating center in the brain are of considerably less importance. This simplifies our problem, as related to the general application of heat, very considerably. There are, however, other problems that are particular to the local application of heat.

The first problem is the fact that increase in the local circulation by the local application of heat is not enough. If, for instance, the size of the blood vessels of the hand has been increased by the local application of heat, this influence will be effective only if the blood vessels leading to the heated area are capable of delivering to that area the increased amount of blood which the increased local circulation requires. In other words, there is no sense in increasing the peripheral circulation if the blood vessels leading up to this peripheral area are not capable of taking care of the increased requirements.

The second basic consideration that confronts us is that in general heating of the body there will be a rise in temperature of the blood which will at least approximately equal that through most of the body. This is not the case in the local heat effect. If the blood gets heated locally by the heating agent, the blood stream will conduct the excess heat away. In other words, normally, the circulation serves as a cooling mechanism of the heated area. This well-known mechanism prevents accumulation of too much heat and the resultant development of burns. This cooling effect of the blood stream then becomes an important addition to the cooling effect produced by local sweating. While in a normal individual this cooling mechanism of the blood stream is a very efficient one, one must realize that, when the normal blood-flow has been impaired, the cooling effect of the blood stream will be nonexistent, or at least severely decreased. This will occur in peripheral vascular diseases, and the danger then will exist of accumulation of large amounts of heat with unfavorable results. This re-emphasizes the well-known fact that an amount of heat which may be well-tolerated in a normal individual can be too much for an individual with impaired peripheral circulation. From this follows the basic consideration that the amount of heat which we can give to a patient, locally, will depend on the seriousness of the involvement of the peripheral circulation.

Another reason why the local heat disappears is the evaporation of sweat. Physiologically, this is an important part of heat loss, and in the intact human body becomes a very important factor. Such secretion, however, is under the influence of the sympathetic nervous system and requires an intact sympathetic nervous system for adequate function. Clinically this is of considerable importance in cases where a sympathectomy has been done, because there is an actual decrease and some-

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times a complete disappearance of the sweat secretion. We all know that sympathectomies are often done in peripheral vascular disease, and the lack of sweat secretion resulting from this type of surgery may seriously impair the removal of heat from the locally heated parts. We then have a situation in which we are dealing with a patient suffering from a peripheral vascular disease who has had a sympathectomy and in whom both mechanisms which remove heat have been interfered with. The blood stream has decreased because of peripheral vascular disease, and the resulting sympathectomy has decreased the sweat secretion, and if the sympathectomy has not been successful in removing the peripheral vascular disease (as it often is) two of the main ways in which excess heat can be taken care of have been considerably decreased or completely eliminated. Obviously the possibility of overdosage of heat becomes a very severe one, and the deeper the penetration of the heat, the greater the danger.

It is obvious from the above that the dosage of heat is not as simple a problem as it is often considered to be. We may consider a moment how the dosage of heat is determined. It is very difficult to measure the amount of heat absorbed by human tissues, except by fairly complicated methods which are not easily applicable clinically. In actual practice the main indication of the amount of heat given is the sensation of the patient. There are two points in this respect with which I think we are all familiar, but which nevertheless seem to be forgotten too often. The first one is a very simple one. If sensation of the patient is the all-important indication of the amount of heat given, it should be a routine procedure in every physical therapy department to test sensation, before any form of heat is given, particularly to an older patient. Many of the patients have involvement of the sensory nerves and as a result our only indicator of heat dosage may not work at all.

The second point of importance in this respect is that the sensation of heat is located in the skin, and that, therefore, for the same amount of heat absorbed by the tissues, the deeper this heat is absorbed, the less the skin sensation will be. In other words, if we give the same amount of heat to the same patient, one in the form of deep heat and the other in the form of superficial heat, the patient will feel considerably less sensation of warmth in the first instance than in the second. It should be realized, for instance, that induction-field diathermy will give relatively less heat sensation than radiant heat. This means, in the first place, that the danger of burns becomes larger, and, in the second place, that the danger of an overdose is considerably greater with the methods which produce more deep heat than with those methods which heat only superficially, such as heat by contact.

Evidences of overdosage of heat are not always recognized to the fullest extent, but one can be aware of them if one is aware of the defense mechanisms of the body against excessive heat. One defense mechanism is an excessive secretion of sweat, another one is a dull, throbbing pain that one so often finds, particularly in the application of too much diathermy. It has been our experience, on careful evaluation, on a clinical basis, of diathermy treatments in the last few years, that, in induction-field diathermy, in many cases all that is required is a lukewarm sensation after an application of twenty minutes, and there have been relatively few instances where more than a sensation of warmness was required after a twenty to thirty-minute treatment.

To refresh your memory for a few minutes on the different types of heat, that is to say, heat by contact, radiant heat, and diathermy, we are all familiar with the fact that, clinically, the main difference between the different types of heat is penetration. Recently, the difference between luminous and nonluminous radiant heat has been studied, and it has been shown that nonluminous infra-red penetrates considerably more deeply than was previously thought possible. Still, if one compares the amount of heating of the skin with the heating of the deeper tissues, the luminous infra-red comes out just as well, and even somewhat better than the nonluminous infra-red. In diathermy it is the induction-field short-wave diathermy, particularly, which produces deep heat, because deeper tissues are better conductors. If one compares the heating obtained by the induction field and by the condensor field, it can, in first approximation, be said that the induction field generally heats the better conducting tissues better than it heats the poorer conductors, while with the condensor field the opposite is true. Industry, which uses both induction-field diathermy and condensor-field diathermy for the heating of materials, is fully aware of this fact. For the heating of insulating materials, such as plastics, industry uses the condensor field; while for the heating of metals in electronic welding and soldering, industry uses the induction field.

In recent years, microwave diathermy has been added to the field of available diathermy wavelengths. Microwave diathermy uses a frequency of 2450 megacycles. Recently other frequencies, namely 9615 megacycles, 5850 megacycles, and 10600 megacycles and 18000 megacycles, have become available for investigation. This is con-

trasted to the three frequencies of induction-field diathermy which are allowed by the Federal Communications Commission, namely, 13.66 megacycles, 27.32 megacycles, and 40.98 megacycles.

Dr. Hines has shown you the physiological evidence that microwave diathermy indeed produces deep heat. As far as the clinical application of microwave is concerned, our evaluation of it is only just beginning. In a recent article by the workers in the Mayo Clinic, they state, "The clinical use of microwaves is only now beginning. However, careful clinical experience, for a period of years, carried out by careful observers, will be required before the clinical status of diathermy with microwave can be definitely established."

There have been some damaging effects of microwave described. Microwaves, particularly, are absorbed by water, and large amounts of fluid can therefore reach extremely high temperatures. On the other hand, a very definite and very deeply penetrating heat can be obtained in a method of application which is very simple.

I think that there is one basic difference between induction-field diathermy and microwave diathermy which will play a very considerable role in the final clinical judgment of the applicability of this method, and that is the size of the area heated. With induction-field diathermy it is very easy to heat a fairly large area with reasonable evenness. The microwave diathermy machine, in its present status, is essentially built to heat rather limited areas. As I have said before, in many cases, where we want to obtain an increase in circulation with our diathermy, we will have to see to it that the blood vessels feeding the heated area are also dilated. In many instances, we will be more interested, clinically, in a heating effect which covers a large area, than in a specifically localized deep heat. On the other hand, the possibility of a well-defined heating area offers clinical opportunities which at present can not be evaluated. It will be particularly worth while to study the combination of microwave diathermy, for a high degree of heat in a circumscribed area, and induction-field diathermy, which would heat a large surrounding area to a lesser degree.

The old-fashioned, long-wave diathermy, with electrodes in direct contact with the skin, can still be used effectively in a number of instances. All of us have treated tendovaginitis of the wrist by a long-wave application between one electrode on the hand and one electrode on the arm. The reason for its effect is that the current concentrates on the area of the wrist's tendons and produces a very mild and beneficial heat in this particular area. Long-wave diathermy is under the control of the Federal Communications Commission, and because of its disturbing effect on broadcasting there is no doubt that any excessive use of long-wave diathermy would result in a speedy effort by the Federal Communications Commission to limit its use.

I think we are all familiar with the contraindications of diathermy. A few of these are specific contraindications of diathermy, others are general contraindications of heat. Among the characteristic contraindications let me remind you of small metal bodies, particularly small pieces of shrapnel in war veterans, also the pregnant uterus and profuse menstruation and bleeding. These are contraindications because they produce large areas of conducting tissue which, therefore, specifically absorb the induction-field diathermy. A similar contraindication is large amounts of fluids, for instance in joints, in the use of microwave diathermy, because it has been shown that microwaves, particularly, are easily absorbed by water. The general contraindications are really based on the previous discussion by Dr. Hines on the physiology of heat, and on what we said before about the balance between the amount of heat introduced and the amount of heat removed by circulation, sweating, and other means. Many of these are, therefore, not absolute contraindications, but are closely related to the amount of heat given. While a small amount of heat is beneficial, a large amount of heat may actually cause serious damage in the form of burns, or in lesser degrees heat may produce a worsening of the original disease. One might express this in the following way:

Heat produces two things, an increased circulation and an increased local process, be it metabolism or disease process or inflammation. And the question is, "Can we increase the circulation to such an extent that the increased circulation can take care of the increased metabolism?" If it can, the balance is favorable, and the applied heat will prove effective. If it cannot, the balance is unfavorable, and we are giving either too much heat, or too deep heat, or we should give no heat at all.

Let me give some specific examples. Nobody would give heat or diathermy in any disease where a flare-up, even a mild one, would be damaging, for instance, in active tuberculosis or malignant tumors. It has often been said that, in arteriosclerosis, diathermy should not be given; this is undoubtedly correct, because, if the arteries are completely stiff and no increase in circulation can occur, diathermy, or heat generally, would hardly be beneficial. All that one would do would be to increase the metabolism and put an increased load on a circulation that cannot increase. On the other

hand, when the narrowing of the arteries is due to spasticity in the walls of the blood vessels, diathermy may cause a very beneficial increase in circulation, and, in this case, the increase in circulation may well offset the increase in metabolism. The actual clinical problem, however, is this—in most cases of peripheral vascular diseases we are not dealing with either complete arteriosclerosis only, or vasospasm only, and will have to evaluate how much of it is due to one factor, and how much of it is due to the other factor, to come to a conclusion as to whether diathermy and other forms of heat can be used effectively. In many instances, the only way to find out is to try it, and thus one will have to be extremely careful and start out with a very mild dosage of diathermy. Only if the mild treatment proves effective, should one very gradually increase the amount of diathermy given. It will be found that a point at which no benefit occurs, and in which a throbbing pain results during the diathermy treatment, will be reached much sooner in these patients than in normal individuals. These actual indications and contraindications for diathermy and heat can be found in any textbook of physical medicine, and I might, for instance, refer you to the very recent article by Martin and Erickson in the new *Handbook of Physical Medicine and Rehabilitation*, or any other good review of medical diathermy or heat.

But, in closing, there are a few points that I would like to mention from our own experience which are based on the above considerations. We are all familiar with the effect of diathermy and deep heat in arthritis, and particularly in osteoarthritis, while most of us would agree that, in rheumatoid arthritis, heat by diathermy is not essential. Partly this is due to the fact that so many joints are involved that it is much easier to treat this patient by short periods of hyper-therm, or in the Hubbard Tank. But the swelling of rheumatoid arthritis contains enough fluid so that overheating of this conductive tissue by diathermy becomes quite easy. When the dosage is reduced, diathermy may become effective.

The particular field which impresses us with the need for very careful dosage is the field of so-called myositis, fibrositis, and bursitis. Since we have watched our dosage of diathermy very carefully, we have generally reduced the amount of diathermy given in these cases, with considerably better results. Now many of these patients are treated in our clinic with an amount of diathermy which produces only a lukewarm sensation after about twenty minutes treatment.

There is one instance in which we have used diathermy which I am quite hesitant to mention. We have used extremely mild diathermy in chronic ulcers due to peripheral vascular disease. Here again in many instances diathermy is obviously contraindicated because a peripheral vascular lesion is entirely due to arteriosclerosis. But in many ulcers there is evidently still enough flexibility of the circulation left for an increase in circulation to be obtained. We have found that a method of choice is a combination of local mecholyl ion transfer to the tissues surrounding the ulcer, together with induction-field diathermy, extremely mild, with a long coil to the entire limb. Obviously, the increase in circulation obtained in this way is very small, but one must not forget that in many instances of an ulcer only a very small increase in vitality of the tissues is needed to start the healing process. Needless to say, the diathermy and the mecholyl ion transfer should not be given simultaneously because the electrodes used for the mecholyl would heat up in the diathermy field. We give the diathermy immediately preceding the mecholyl ion transfer.

The emphasis on mild diathermy brings up a point in Dr. Hines' discussion which appears to be of major importance. Dr. Hines has shown, and has emphasized here, that, in order to get vasodilatation, a certain definite increase in temperature must be obtained. It is likely, if not certain, that many of our clinical applications are below this threshold. One would then ask the question, how can the effect of diathermy be explained, if, in some cases, it is not enough to cause appreciable vasodilatation. Dr. Hines has also emphasized that exercise, that is rhythmical muscle contractions (not sustained ones), causes a greater degree of increase in circulation than most methods of local heat application; yet, clinically, one would not want to suggest the use of exercise as a replacement for local heat. Here again the question should be raised, whether or not local heat can perform its useful function in some other way than by vasodilatation. Obviously this problem does not involve any specific effect of any one of the methods capable of producing heat. The question is—what is the mechanism of the beneficial effect of local heat? Is it vasodilatation only, or are other factors involved? Dr. Hines has pointed out a number of these factors. It will require much future research to evaluate these and other possible factors.

The importance of this for the clinical application of local heat is, of course, obvious. Only if we understand *how* our local heat produces its effects can we come to a really intelligent application of this important physical modality.

The Role of Circulation in the Physiology of Heat Regulation

Allen D. Keller, Ph.D.

Regulation of Body Temperature

In heat-regulating animals the temperature of the central core of the body is maintained at a relatively constant level, regardless of a wide range of environmental temperatures to which the animal may be exposed. This can be demonstrated conveniently in the laboratory, using the dog as the experimental animal.

When a dog is abruptly removed from a laboratory temperature of 23-24° C. and placed into a refrigerated room held at −20° C., its rectal temperature actually rises somewhat, where it may be maintained for the duration of the experiment, or it may return progressively to or toward the pre-exposure level. This is illustrated in Figure 1a. In this particular experiment the rectal temperature rose from 38 to 38.5° C. in the first thirty minutes, and to 38.7° C. at the end of the first hour. It was maintained at this level for the duration of the twenty-four-hour exposure. Thus the rectal temperature was maintained at slightly above its usual level against a definitely heavy external cooling load.

Again, if a dog is suddenly subjected to an

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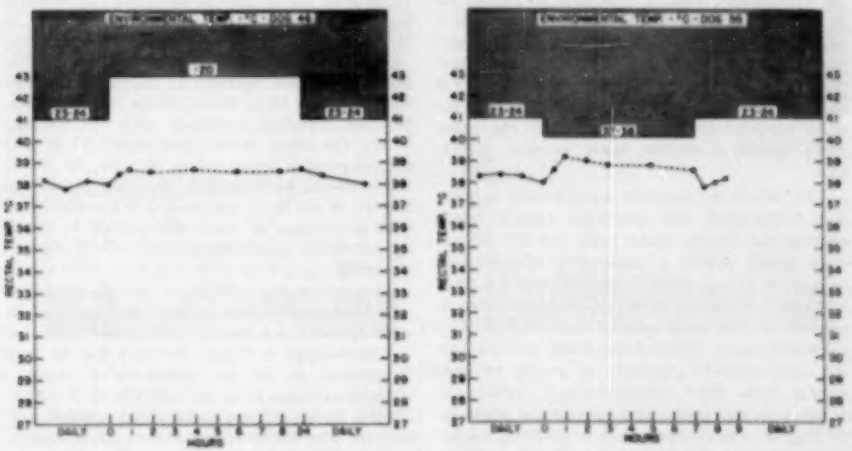

Figure 1.

a. Rectal temperature curve of an unoperated dog during a twenty-four-hour exposure to an environmental temperature of −20° C. Stippling in the curve indicates the presence of shivering.

b. Rectal temperature curve of an unoperated dog during a seven-hour exposure to an environmental temperature of 37-38° C. Stippling in the curve indicates the presence of panting.

Figure 2. An artist's drawing of the medial half of a dog's brain upon which is indicated the location of the heat-regulating centers. The area circumscribed by the solid line is the approximate amount of tissue which must be destroyed to eliminate an animal's ability to regulate against a cool environment. The area circumscribed by stippling is the approximate extent to which heat-loss cells extend into the cephalic midbrain.

environmental temperature of 37-38° C., the rectal temperature rises to a slightly higher level, where it may be maintained, but it usually returns progressively toward the pre-exposure level during the experiment. This is illustrated in Figure 1b. In this particular experiment the rectal temperature rose from 38 to 38.6° C. in the first 30 minutes and to 39.2° C. at the end of the first hour. From this level it decreased gradually, being 38.6° C. at the end of the seven-hour exposure. Thus the rectal temperature was not allowed to rise excessively against a definite heavy external heating load.

This ability to maintain automatically a body-core temperature near constancy, against drastic cooling and heating loads, holds true for all animals which possess a reasonably efficient heat regulation. (Dog, monkey, ape, and man are representative.) It should be appreciated that different animals of the same species may react slightly differently under identical conditions and also that the same animal's responses to graded environmental loads show correspondingly minor differences. In this connection compare the responses of Dog 46 under cooling loads of different magnitudes (shown in Figures 1a and 3a). These variations in response are relatively unimportant; the important point is that a frank hypothermia or hyperthermia is not allowed to develop. It is this end-result which is reliably predictable.

Heat Regulating Centers or Thermostats

The maintenance of a body temperature near constancy is the result of regulatory processes mediated by the central nervous system. The responsible, central, integrating mechanisms are located in specific areas in the brain stem, as schematized in Figure 2.

The center or thermostat which regulates against a cool or cold environment is confined to that portion of the brain stem known as the hypothalamus. Although there is reason for believing that nerve cells responsible for regulation against a warm or hot environment are also located in the hypothalamus, it is known that some of these cells, sufficient in number to maintain adequate heat-dissipating functions, reach caudally into the adjacent midbrain. When these areas are destroyed by appropriate surgical procedures, the animals lose their respective abilities to maintain a constant core-temperature against adverse environments; they are reduced to non-heat-regulating preparations.

An experimental animal (taken from our operated series), which suffered a complete loss of ability to regulate against a cool environment (Dog 28), is illustrated in Figure 3a. After operation it was necessary to house this animal in an environment of 30-31° C. to keep its rectal temperature in the neighborhood of 38° C. When it was abruptly placed in an environment of 5° C. its rectal temperature fell progressively, and reached 28° C. at the end of a three-hour exposure. The animal was then removed to an environment of 22-23° C. for three hours, where the rectal temperature remained stationary with no tendency to rise. On return to the warm room (30-31° C.) the temperature began rising, returning to 38° C. the following morning. It is important to note that the fall of the rectal temperature is a straight line, with no changes in slope which could be caused by remnantal regulatory powers. There was no shivering.

In spite of the permanent loss of all central cold-combating powers in Dog 28, its heat-dissipating abilities remained undisturbed! This fact is demonstrated in Figure 3b. Note that its rectal temperature did not rise beyond 39° C. during a six-hour exposure to an environment of 37-38° C. Typical slow and easy panting was present continually, after the rectal temperature reached 39° C. It is to be emphasized that in this dog *the thermostat which guards against a lowering of body-core temperature was selectively destroyed without impairing in the least the thermostat which regulates against a hot environment.* Such a dog,

running at large in Miami, would probably survive the seasons and die of old age, but, in Cleveland, with the onset of winter it would surely die from the effects of hypothermia.

When the heat-dissipating thermostat is destroyed and the animal is subjected to a hot environment, the rectal temperature rises rapidly, and the animal will die of heat prostration unless the exposure to heat is ended shortly after the rectal temperature reaches 43° C. Such a response is also illustrated in Figure 3b. Note that in this dog (72-D) the rectal temperature rose from 36 to 43° C. in an hour and a half. The slope of the temperature curve actually steepened with time. This was, of course, partly due to increased tissue metabolism caused by a rise in temperature, but it was also accentuated by increased activity, e.g., walking rapidly about cage. There was no panting.

PERIPHERAL PHYSIOLOGICAL CONTRIVANCES UTILIZED IN HEAT REGULATION

The foregoing experiments demonstrate: first, what is meant by the term *heat regulation*, and, second, that the maintenance of a constant body-core temperature is dependent on specific *neurogenic thermostatic controls*. Now the question arises, how do these central neurological mechanisms attain constancy of body-core temperature? Or, stated in another way, what peripheral physiological contrivances are utilized by these central integrative mechanisms?

The individual physiological processes known to be, and suspected to be, utilized in this connection are numerous. But, as we shall demonstrate later, it is difficult to isolate and evaluate physiologically the exact load carried by any one individual peripheral reaction. It is also difficult

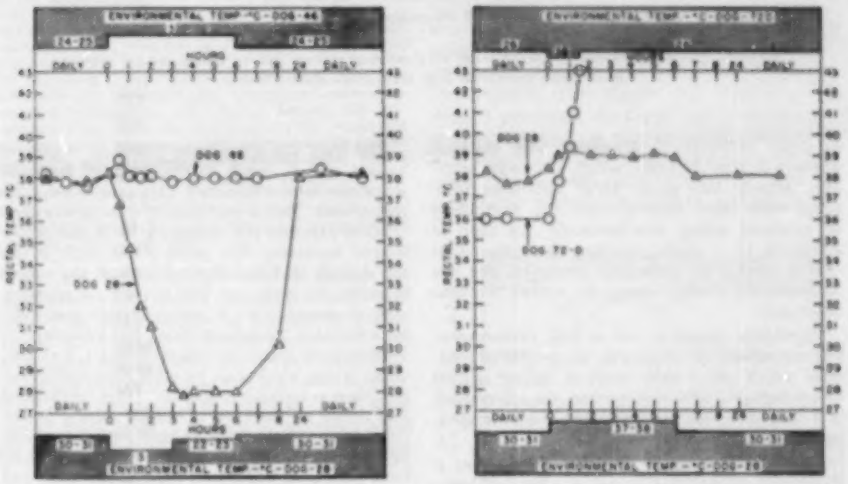

Figure 3.

a. Rectal temperature curve of a dog (28) in which all central regulation against a cool environment was eliminated by a large lesion placed in the posterior hypothalamus. This dog's temperature curve is contrasted with an unoperated dog's (46) temperature curve, when stimulated by the same cooling load. Stippling in the curve indicates the presence of shivering.

b. Rectal temperature curve, of the same operated dog shown in "a," during a six-hour exposure to an environmental temperature of 37-38°C. This dog's entirely normal regulation against a heavy heat-load is contrasted with the rectal temperature response of another dog (72-D) in which all heat dissipating abilities were absent. Stippling indicates the presence of panting.

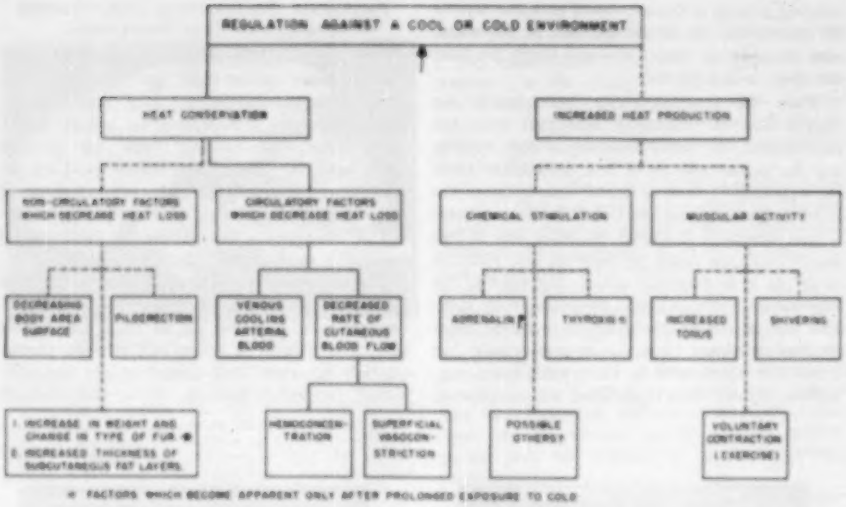

Figure 4. Schematic diagram of the processes involved in regulation against a cool or cold environment. Solid connection lines indicate circulatory factors.

in many instances to determine with certainty whether certain reactions, which are under suspicion, actually take place. These difficulties arise, along with other factors, from the multiplicity of processes acting simultaneously, the time of activation of a given reaction, the tremendous reserve present in individual processes, and the compensatory overlap among the various reactions concerned.

Regulation against a cool or cold environment. For convenience of discussion, the peripheral processes which are usually credited to be utilized in combating a cool environment are schematized in a simplified, but not necessarily comprehensive, form in Figure 4.

When a lowering of the body temperature is threatened, the problem is met in a twofold manner. First, an effective physiological effort is made to prevent the heat produced under ordinary conditions from escaping from the body (heat conservation outflow). Second, when additional heat is needed, it is produced by stimulating heat-producing processes to greater activity, and by activating reserve contrivances specifically designed or adapted for heat-producing purposes (heat production outflow).

The part the circulation plays in regulation against cold is clearly in the realm of *heat conservation;* here, circulatory adjustments carry the major load. This is particularly true during short bouts of exposure and during the early part of prolonged exposures. The primary end-result is that the amount of blood flowing through the surface layers of the body, per unit of time, is appropriately decreased. This is brought about principally by cutaneous vasoconstriction (accompanied by compensatory splanchnic vasodilatation), and, also, under certain conditions, by other contributing factors, such as hemoconcentration. These conclusions, first drawn on the basis of simple clinical inspection, using criteria such as skin color, skin temperature, and increased difficulty in drawing blood from peripheral veins, have subsequently been supported by more exact laboratory analyses.

It has been shown that there are quantitative limitations to the use of skin temperature as an indication of blood flow. However, certain of these limitations are readily appreciated, and they do not invalidate the use of the skin temperature, in many circumstances, as a reliable and useful qualitative index of blood flow.

There are several different ways of demonstrating that reflex cutaneous vasoconstriction occurs as a direct result of a peripheral cold stimulus. An experiment of Doupe *et al.* is particularly clear-cut and convincing. They ex-

posed an eight-centimeter length of a wrist vein in man, closed it off temporarily at both ends, and, by means of a hypodermic needle, emptied it of blood and refilled it with saline. Then, by connecting the needle with a pressure recording apparatus, they demonstrated a vigorous contraction of the segment of vein when cold was applied to the surface of the body.

The suspicion that hemoconcentration may occur under certain circumstances of cold exposure is given strong presumptive support by an experiment on our Dog 26. The results and the nature of the experiment are recorded in Figure 5. This dog's ability to combat a cold environment had previously been partially impaired by a moderately large lesion placed in the posterior hypothalamus. Thus, as will be seen in the figure, when it was subjected to an environment of 5° C. for several days, the rectal temperature dropped from the normal range centering around 38° C. to a lower range centering around 35° C. and the hematocrit rose considerably. Both of these changes were maintained for the duration of the exposure. It should be appreciated that the hemoconcentration in this instance was out of proportion to what might occur in the unoperated animal, because of the partial impairment in its ability to regulate against cold.

An insight into a factor in the mechanism of attaining hemoconcentration is also illustrated in this experiment. The dog's water consumption was strikingly decreased at the same time that the hematocrit was elevated. In fact, in order to get the dog to take any fluid at all, it was necessary to bait it with food, by adding a small amount of milk. Note that on the fourth day, when the dog was given a greater amount of fluid in this manner, the hematocrit showed a corresponding drop. Also note that both the hematocrit and spontaneous water consumption returned to, and slightly beyond, the pre-exposure levels when the cold exposure was terminated and the animal was housed in a warm room. In connection with this experiment, one should recall that a diuresis is frequently, if not usually, precipitated by a cold stimulus in humans. It has also been shown that, at least in instances, there is associated with "cold diuresis" a maintained loss in body water during a semiprolonged exposure to moderate cold.

But how does reducing blood flow through the skin conserve body heat? It does so in two different ways. First, the blood passing through the surface vessels serves directly as a fluid transport of heat. It becomes ladened with heat in the deep heat-producing organs and loses it to the cooler environment during its passage through the surface tissue layers. Accordingly, a decrease in cutaneous blood flow means less loss of body heat from direct fluid transport to the body surface. Second, a decrease in peripheral blood flow increases proportionately the insulating properties of the cutaneous and subcutaneous tissue layers. For example, it has been estimated that a subcutaneous layer of fat, free of any blood flow, has as much insulating capacity as a layer of cork of the same thickness. Thus, insulation in the body wall of a heat-regulating animal, by virtue of blood flow, is a dynamic, automatic, adjustable affair, not a static affair such as is found in the walls of a laboratory refrigerator or oven.

To demonstrate that a certain peripheral process is activated, during exposure of an animal to adverse environmental loads, is one thing, but to determine its relative isolated importance in maintaining a constant body temperature is an entirely different matter. For instance, there is no doubt that a vigorous cutaneous vasoconstriction occurs as a result of exposure to cold. On this basis it would be reasonable to assume that the maintenance of a constant body-core temperature against a cooling load is, to a large degree, dependent upon the central activation of this single peripheral reaction. Nevertheless the question remains, to what extent would an animal's ability to prevent a lowering of body temperature actually be impaired by removal of all central vasoconstrictor control? This approach (the determining of physiological deficits) is a most important one, an analytical approach which should never be neglected. It can be used experimentally, with respect to the central vasoconstrictor control, in a way which is reasonably free of complications. The entire outflow of vasoconstrictor nerve fibers is severed by surgical extirpation of the thoracic and abdominal sympathetic chains. *(See Figure 6.)* Following this surgical procedure, the blood vessels become permanently dilated, as can be readily attested by such simple criteria as direct visualization of cutaneous blood vessels, skin temperature, and the greater ease in obtaining blood by venipuncture.

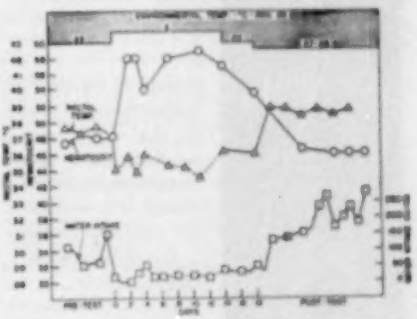

Figure 5. Rectal temperature, hematocrit, and water-intake curves of Dog 26 before, during, and after exposure to a 5° C. ambient temperature for fourteen days. Stippling in the water-intake curve indicates that a minimal amount of water was baited with milk. See text for further details.

Such a sympathectomized dog maintains a rectal temperature within the normal range of 38°C. when it is housed at ordinary laboratory temperatures. Even when it is subjected to a heavy external cooling load, the magnitude of the hypothermia which develops is surprisingly small! These facts are shown in our experiment on Dog 46 and illustrated in Figure 7. In this particular experiment, the dog was subjected to an environmental temperature of 5° C. for six hours. The rectal temperature fell slowly during the first four hours, reaching 35.2° C. During the succeeding two hours, the rectal temperature actually began to return to the pre-exposure level. Thus, in the complete absence of central vasoconstrictor control, a serious hypothermia did not develop. This seems almost incredible; by comparison with a mechanical model, it amounts to keeping the temperature of the interior of a house on a reasonably even keel on a very cold day, when the house has more than half of the exterior wall space completely open-windowed.

This essentially adequate regulation against cold in the presence of bilateral sympathectomy, shown in the foregoing experiment, is an example of: first, the tremendous reserve of regulating contrivances (compensatory processes) possessed by heat-regulating animals, and, second, the difficulty of evaluting quantitatively the role of a single individual peripheral reaction in the over-all heat regulatory mechanism.

Figure 6. Photograph of the sympathetic chains of Dog 46 after surgical removal and fixation in 10 percent neutral formalin. Successful removal of each chain in one piece gives assurance of complete removal.

A specific compensatory factor, which should be mentioned in this connection, is that considerable vasoconstriction occurs in the absence of central vasoconstrictor control, due to cold acting directly on the blood vessels. That such is the case is readily evident in these preparations by the increased difficulty of drawing blood from a peripheral vein during a bout of cold exposure; whereas in the same dog, previous to, or after a cold exposure, this is an easy matter because of the chronic vasodilatation.

Regulation against a warm or hot environment. The peripheral contrivances which are known to be utilized in the central regulation against an environmental heat-load are schematized in Figure 8. The problem of dissipating excess body heat is met first, by increasing the blood flow through the surface layers of the body, and second, by activating devices specifically designed or adapted for heat-dissipating purposes (sweating or panting). During exposure to a mild or moderate heat load, heat dissipation is achieved largely by circulatory adjustments. During exposure to heavy heat loads, these circulatory adjustments continue active, and may be intensified. In addition, the blood supplies the sweat glands and/or lungs with the necessary fluid for the reserve evaporative cooling which is achieved through sweating or panting.

The primary end-result of the circulatory adjustments is an appropriate increase in the amount of

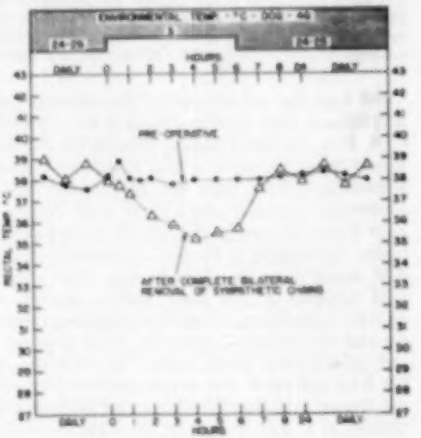

Figure 7. Preoperative and post-sympathectomized rectal temperature curves of Dog 46 during exposure to an ambient temperature of 5°C. for a six-hour period.

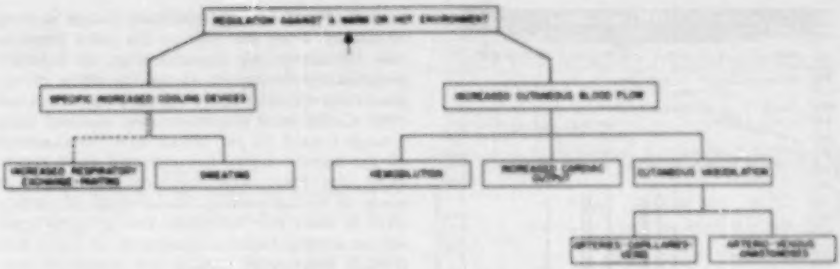

Figure 8. Schematic diagram of the processes involved in regulation against a warm or hot environment. Solid connection lines indicate circulatory factors, directly and indirectly.

blood flowing through the surface layers of the body per unit of time. This is brought about principally by cutaneous vasodilatation (accompanied during the early part of the exposure by compensatory splanchnic constriction), but other adjustments, such as increased cardiac-output hemodilution and increased circulating blood-volume, become apparent with time. The cutaneous vasodilation is readily apparent on the basis of simple clinical inspection. In the dog, for instance, the skin on the parts of the body where the hair is thin is visibly pink. The temperature of the skin is obviously elevated above what obtains at ordinary room temperatures, and a large quantity of blood can be drawn from a leg vein with greater ease than under ordinary circumstances. In addition, blood drawn from a vein, when an animal is actively combating a moderately warm or hot environment, looks like arterial, rather than venous, blood. This means that more blood is passing through the tissue which the vein drains than is necessary to supply the oxygen requirements of the tissue. This is direct evidence of an increased blood flow. Thus one does not need exacting laboratory analyses to be convinced that, under these conditions, the dilatation of the superficial veins in the limbs is associated with a fast and/or increased cutaneous blood flow.

An interesting anatomical device has recently been described which is utilized for increasing the blood flow through the skin, apparently for specific heat-dissipation purposes. This anatomical contrivance is the arteriovenous anastomosis. By these anastomoses, blood is shunted past the capillaries, from the arteries, directly into the veins. The blood so shunted has no opportunity of having any oxygen extracted from it by the tissues.

How, then, does this increase in blood flow, through the surface layers of the body, facilitate the dissipation of excess body heat? It does so in two definite ways, being the exact opposite of what occurs as a result of decreased flow during exposure to cold. First, an increase in peripheral blood flow means that this blood can lose heat to the environment, directly and indirectly, by fluid transport from the deep heat-producing organs, just as the hot water circulated through an automobile engine loses heat to the environment as it passes through the radiator. Secondly, an increase in peripheral blood flow decreases proportionately the insulating properties of the cutaneous and subcutaneous tissue layers; in terms of a mechanical model it is somewhat equivalent to removing the rock wool from the exterior walls of a house and perhaps, at the same time, puncturing a good many holes in the remaining wall structure.

It is common experience that most people do not begin sweating until the weather becomes quite warm. Thus, sweating in man and panting in the dog are reserve body-cooling devices which ordinarily are not activated until the heat load becomes distinctly obvious. This observation, when analyzed, furnishes evidence in support of a previous statement—"during exposure to moderate, environmental heat-loads, adequate heat dissipation is achieved largely by circulatory adjustment." By subjecting a dog to graded ambient temperatures, evidence in support of this statement became even more convincing, e.g., on a qualitative basis. The adult dog has no sweat glands of any consequence, and it achieves evaporation cooling through the mechanism of an increase in lung ventilation. The magnitude of the latter can be estimated directly by counting the respiratory rate and noting the presence or absence of panting. When one has a quiet dog, the magnitude of the environmental heat-load which can be tolerated without activating panting can be determined with relative ease.

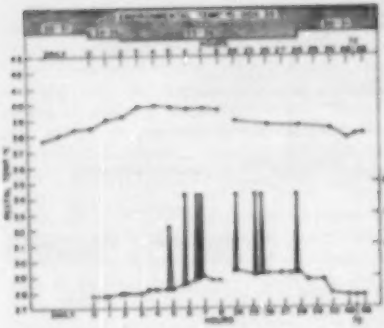

Figure 9. Rectal temperature and respiratory rate curves of Dog 35 during a twenty-eight-hour exposure to an ambient temperature of 35-36° C. See text for further details.

An experiment on Dog 35, from our series, illustrates this point; the relevant data being charted in Figure 9. This dog is the same type of preparation as Dog 28, described previously. By virtue of a large posterior hypothalamic lesion, all ability to regulate against a cool or cold environment was permanently eliminated, but, as is evident in Figure 9, his regulation against a moderately heavy heat-load was not only adequate, but remained entirely within the normal response range. This animal was decidedly on the quiet side; he would lie still for hours, moving only to eat, drink, urinate, defecate, and occasionally to change position. When the cage was approached he would raise and turn his head, watch the approacher carefully, and, if the occasion seemed to warrant it, would growl furiously, with associated piloerection. Fortunately, these spurts of rage did not occur during heat tolerance tests, and therefore internal heat production remained on a relatively even keel.

In this particular experiment (as in the case of Dog 28), it was necessary to house the animal in an environment of 30-31° C. in order to keep the rectal temperature around 38° C. When it was abruptly placed in an ambient temperature of 34-35° C., the rectal temperature rose from 38.5 to 39° C. in one hour, where it leveled off, being only 39.2° C. at the end of the second hour. This leveling-off occurred in the absence of any increase in lung ventilation. The ambient temperature was then raised to 35-36° C. At the end of the first hour at this temperature, the rectal temperature reached 39.8° C., and, after the second hour, it was 39.9° C. Here again the rectal temperature leveled off without any significant change in lung ventilation. From this time on the rectal temperature did not go any higher, rather, it decreased progressively during the succeeding hours of exposure. Shortly after the rectal temperature reached 39.8° C. the basal respiratory rate increased from a range around 20 per minute to a range around 60 per minute. Upon this increased basal respiratory rate there were superimposed occasional bursts of typical panting. These bursts of panting were so short and infrequent that it seems quite safe to assume that the dissipation of body heat through evaporative cooling was essentially negligible in this experiment.

An interesting sidepoint is that these bursts of panting were definitely not elicited directly by heat stimuli, ambient temperature, or body temperature, but were clearly activated by movement of the body or a part of the body. Thus, when one approached the experimental box, the dog would raise and turn his head or get on his feet; immediately, panting would begin, but it would stop abruptly when the animal settled down. Obviously, receptor organs were activated due to movement in muscles or joints, which set off evaporative cooling in anticipation of an increased internal heat production, rather than as a result of such an added internal load.

A point of practical significance is the fact that the efficiency and ease in regulating against a warm or hot environment increases rapidly during the early part of an exposure. Note from Figure 9 that the rectal temperature in Dog 35 had begun dropping slightly at the end of the third hour of the 35-36° C. exposure and that this drop was considerable between the eighth and the twenty-fourth hour, having decreased from 39.6 to 38.9° C. Here again, it seems safe to assume that this progressive adaptation to the external heat load was brought about principally through circulatory adjustments; certainly, evaporative cooling through panting did not play an appreciable role. There is evidence in the literature that, clouded in most instances by intermittence of exposure and superimposed effects of exercise, a predominant, responsible, circulatory adjustment in adaptation to heat is a progressive increase in circulating blood volume, with the re-establishment of pre-exposure concentration of the various blood constituents. This presumably relieves certain early compensatory responses, such as splanchnic vasoconstriction and a raised arterial pressure, and at the same time makes possible an even greater cutaneous vasodilatation and increase in peripheral blood flow.

Summary

During exposure to moderately adverse environments, heat conservation or heat dissipation, as the

case may be, is achieved by circulatory adjustments, or largely so.

When the environmental load threatens the capacity of circulatory adjustments, non-circulatory, reserve heat-producing or heat-dissipating devices, as the case may be, are activated to meet the excessive load.

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Contributors to the Symposium

Bouman, Harry D., M.D.—Professor of Physical Medicine, University of Wisconsin Medical School, Coordinator of the Physiology Symposium for the 1950 Conference.

Dr. Bouman has held numerous positions in basic and clinical physiology.

He has held a Rockefeller Foundation Fellowship at University College, London, and a Stokvis Foundation Fellowship at the Sorbonne of Paris. He has been a fellow in physiology at Washington University, St. Louis, and a fellow in physiology and orthopedics at the University of Rochester.

Dr. Bouman has also taught physiological psychology at Oberlin College and at the University of Rochester. He was Assistant Professor of Physical Medicine at Northwestern University Medical School before moving to his present position in 1947.

He is a member of the American Physiological Society, the Society for Experimental Biology and Medicine, and is a Fellow of the American Psychological Association. He has done both basic and clinical research in the fields of physiology of muscle and nerve, the sense organs, and biophysics.

Hines, Harry M., Ph.D.—Professor and Head of Physiology Department, State University of Iowa Medical School.

Dr. Hines received his B.A., M.S., and Ph.D. from Iowa. He has served as Instructor, Assistant Professor, Associate Professor, and Professor at Iowa, and Research Associate, Cornell Medical College.

Dr. Hines is a member of many professional societies and received the Gold Medal of the American Congress of Physical Medicine in 1946 and 1949. He has published and done research on many subjects, for example, neuromuscular atrophy and regeneration, muscle chemistry, and muscular dystrophy.

Keller, Allen D., Ph.D.—Chief, Physiology Branch, and Chief, Section of Central Nervous System Physiology of the Medical Department, Field Research Laboratory, U. S. Army, Fort Knox, Kentucky; Lecturer in Physiology, School of Medicine, University of Texas; Professorial Associate in Physiology, University of Louisville, School of Medicine.

Dr. Keller was born in Idaho. He received his B.S. degree from the Utah State Agricultural College and his Ph.D. from Cornell University.

Since 1925 he has been continually active in medical teaching and research, concentrating on research on the physiology of intracranial organs, with particular attention to the neurological regulation of body temperature.

Randall, Barbara Feucht, M.S., R.P.T.—Research Assistant in the Department of Physiology, College of Medicine, State University of Iowa, while doing predoctorate studies.

Mrs. Randall received her B.S. from State Teachers College, Cortland, New York. She received her physical therapy training and her M.S. degree from the State University of Iowa.

She is a member of the American Physical Therapy Association, the American Registry of Physical Therapists, and the Sigma Xi.

She is the author of papers on blood flow and on the effect of metal implants on heating with diathermy.

Wise, Charles, M.D.—Professor of Physical Medicine, The George Washington University School of Medicine; Director, Department of Physical Medicine, The George Washington University Hospital; Attending Physician in Physical Medicine, Mt. Alto Veterans Hospital, Washington, D. C.; Consultant in Physical Medicine, Walter Reed General Hospital.

Dr. Wise received his B.S. and M.D. from New York University. He served in the Army Medical Corps and later was research Fellow in Physiology, Harvard Medical School, and Baruch Fellow in Physical Medicine, Harvard Medical School.

Dr. Wise is a member of the American Congress of Physical Medicine; the Consultants on Education, Council on Physical Medicine and Rehabilitation, American Medical Association; and a diplomate of the American Board of Physical Medicine and Rehabilitation.

Editorials

In this issue will be found the second series of papers presented in Cleveland as part of the "Symposium on Physiology." For your convenience, this issue is published at an earlier date than usual in order to bring the entire "Symposium" to you at one time.

It is our hope that this material will prove of value to all physical therapists. Since we anticipate that many of you will wish additional copies, this material will be published in book form in its entirety. Details of price and date of publication will be published soon.

You are reminded that much of this material contains observations and results from original work and is not available in any other form. This information was selected and presented with the physical therapist in mind and has been compiled in order to assist each one of us to understand better the physiological principles underlying effective patient treatment. It is sincerely hoped that it will prove to be a valuable addition to your professional library.

The Editorial Board of the Review will welcome your comments and suggestions regarding this material.

The Director Reports:

October was a field trip month for the Director —Philadelphia for a meeting of the Advisory Committee to the Educational Consultant, consisting of Florence Linduff, Jane Carlin, and Dorothy Baethke, followed by a chapter meeting. Plans for the 1952 Conference were also initiated. Then across country to Omaha, Nebraska. Clinical facilities were visited there, in Lincoln, and in Council Bluffs, Iowa. Enid Bailey, President of the Nebraska Chapter, had a well-planned itinerary which included a luncheon with the members in Lincoln and a luncheon with the members in Omaha, including a member of the Chapter's Medical Advisory Council in both cities. It was both pleasant and profitable.

Colorado and the American Occupational Therapy Association's Annual Conference at Glenwood Springs was next. As you know, the American Physical Therapy Association's Annual Conference is to be held there June 17-22, 1951. The weather was ideal, unusually warm and sunny, with a refreshing coolness in the evening. Marian Williams and Olena Cole, of our Conference Program Committee, met there to make plans for next June. Colorado committee members, Esther Gillette, President, and Evelyn May, came over from Denver to discuss general plans. It is an ideal spot for a conference and vacation. The whole hotel will be ours. Rates are reasonable and are based on American plan: eleven dollars a day for two in a room; ten dollars for three in a room. There are no singles. The rooms are large, with very comfortable beds.

You will hear more from time to time of all the exciting extra-curricular activities, plus a very stimulating program. The Colorado chapter is going "all out" to make this conference a memorable one.

Other physical therapists at Glenwood Springs included Catherine Worthingham, Eleanor Loomis, Major Underkoffler, Marion Easton, and Margaret Rood. The latter two are also occupational therapists.

In Denver there were further enthusiastic meetings on conference. Sixty-two attended a dinner meeting and I guarantee that this conference will be one you will not wish to miss.

In Chicago there was the Annual Convention of the National Society for Crippled Children and Adults. Many physical therapists were attending the sessions and were at the luncheon for physical therapists and occupational therapists. Catherine Worthingham was on a panel, "Mobilizing Personnel for Service." The shortage of personnel was emphasized. Louise Bailey, a Director of the Association, and Consultant in Cerebral Palsy for the National Society, was among the busiest people there. The program was uniformly excellent.

The Illinois chapter invited me to an executive and advisory committee meeting which was most stimulating. One of their biggest projects is legislation. An unusual departure from their regularly planned meetings is a Sunday brunch on November 5. It is hoped that this will bring in many who cannot get to evening meetings because of distance.

In the National Office work continues at a merry pace. Large numbers of the brochure are being mailed. Keep plenty on hand in your chapters

for we may get some mid-term classes started and they must be filled. Assistance for you in a department depends on your recruitment.

There is still a high polio incidence and there are many requests for emergency personnel which cannot be filled. Every effort must be made to work out ways and means of caring for these requests locally.

Margaret Moore was a participant on a panel at the Military Surgeons' meeting in New York on November 9. Her topic was "There Are Challenges in Electrotherapy."

Mary Haskell and Floy Pinkerton attended a Connecticut chapter meeting on October 17.

The Association was represented at the Mid-century White House Conference on Children and Youth, December 3-7, by Mary Clyde Singleton, President.

The semi-annual Executive Committee meeting was held in New York, November 30, and December 1 and 2. In a future REVIEW the Secretary will summarize the action taken.

—MILDRED ELSON

World Confederation for Physical Therapy

The date given for the World Confederation for Physical Therapy in the October REVIEW is incorrect. It will be September **7** and **8** *instead of* September 1. This will permit attending physical therapists to go directly to Stockholm to attend the International Council for the Care of Cripples in Stockholm, September 9-15, and to attend the Second International Polio Conference in Copenhagen, September 3-7.

It is hoped that physical therapists throughout the world will take advantage of this unusual opportunity to attend and be a part of the organizational meeting of the World Confederation for Physical Therapy.

The programs of the Second International Polio Conference and the International Council for the Care of Cripples will include physicians and scientists from many countries. There will be exhibits and demonstrations of special interest to physical therapists. Details of the physical therapy program will appear in the REVIEW and will be sent to physical therapy associations throughout the world in January.

Arrangements for housing are being worked out and definite plans will be announced later.

Information about travel arrangements for those attending from Canada and the United States will soon be available.

Plan now to attend.

Membership News

Marriages

Lois L. Garraway of St. Louis, now Mrs. Lois L. Taber of Arlington, Va.

Elizabeth Kimbro, Salt Lake City, now Mrs. Elizabeth K. Mooney.

Jean Kennedy, Pennsylvania, now Mrs. Jean K. Block.

Jeanne Casas, San Francisco, now Mrs. Jeanne C. Tomasello.

Vivian Alvey, Wausau, Wis., now Mrs. Vivian Urmanski.

Lois Mae Boldt of Appleton, Wis., to Julius Kopplin, Jr., Highland, Ill.

Patricia Moir of Monrovia, now Mrs. Patricia M. Kallen, Sierra Madre, Calif.

Chessie Zhikowska of Salem, Mass., to George Kouba, Urbana, Ill.

Mary Bickel, Omaha, now Mrs. Mary Lindhall.

Virginia Donvanco to Robert W. Bauer, San Francisco, October 9th.

Joyce C. White to Henry A. Fredrickson, Detroit.

Margaret A. Van Wart, Swoyckley, Pa., to Michael Beyda, Jr., Wallington, N. J.

Patricia Yoder of Madison, now Mrs. Patricia Y. Quin, Green Bay, Wis.

Alice H. Smith, Schenectady, to David L. May, Brewerton, N. Y., September 9th.

Jean Woelfer of Madison, Wis., now Mrs. Jean B. Humphreys, Middletown, Conn.

Mary Jane Watson of San Mateo to Robert J. Redeker of San Jose, Calif.

Frances Canavan of Vancouver, B. C., to Gordon Cruise, Vancouver.

Gillian F. Griffith, Durham, N. C., to Stephen B. Smith.

Jacquelyn Peterson of New Richmond, Wis., now Mrs. Jacquelyn Reeve of Schenectady, N. Y.

Betty Jo Lawless of Camp Point, Ill., now Mrs. Betty Jo Seifert, Modesto, Calif.

Mildred Rosen, Brooklyn, now Mrs. Mildred R. Rose.

Elizabeth W. Hall, Fargo, N. D., now Mrs. Elizabeth H. McLaughlin of Moorhead, Minn.

Jeannette Horton of Rocky River, Ohio, to Homer Hollar, Wilmington, N. C.

Joyce Ryan, Hartford, Conn., now Mrs. Joyce R. La Motte.

Jane Walker of Manchester to Lawrence B. Swallow of Portsmouth, N. H.

Buena M. Stewart of Salem, Ore., to Frank M. Washburn, Springfield, Mass.

Sally A. Fletcher of Shrewsbury to Robert L. Hellens, Princeton, Mass.

A. Fern Green to Luther Livingston, Los Alamos, N. M., October 12th.

Jean Harris, Watertown, now Mrs. Jean H. Archbald, Buffalo, N. Y.

New Members

The following are newly accepted members of the American Physical Therapy Association:

Justin Alexander
Bertha Andrews
Paul T. Bailey, Jr.
Margaret Elizabeth Bacon
Mary Louise Brown
Genevieve Colvin
Patrick L. Cozzo
Harry Edward Dean, Jr.
Helen M. Dobbin
Zora Jane Rhodes
Barbara Ellen Ferguson
Gloria Joan Fitzpatrick
Mary Elizabeth Graves
James W. Herrington
Joe Harold Holler
Robert C. Holmes
Andrew W. Kotsopoulos
Amalia Helen Krajewska
Charles Leudecke
Thomas J. Marchigiano
Viola McCormick
Merton Edward Miller
John S. Naspinsky
Cecile Hope Pate
James F. Peck
Sara M. Raymer
Cecelia Eve Rubnitz
Betty Burt Saunders
Paul J. Ward
Lillian R. Webber

New Student Members

Baruch Center of Physical Medicine of the Medical College of Virginia

Minnie D. Banko
Elizabeth Campbell
Mary E. Campbell
Nelson Cornea
Regina Crawford
Marion M. Dooley
Betty Jane Edwards
Everett Gruelle
John L. Hall
Ivan C. Kline
Jamie L. Lisle
P. Jane McNaughton
Richard Magano
Charlene Nelson
Margaret Wells
Mrs. Shirley M. Wood
Evangeline Y. Zehmer
Angela L. Colon
Pauline F. Bargion
Johnnie R. Ellis
Frances M. Springer
Sara Jean Coughenour
James H. Poulson
Alice L. Quinn
Bernard W. Swift
Alfred J. Szumski
Mary M. Urban

Boston-Boston School of Physical Education

Anne Ainsworth
Nancy Allott
Anne Cody
Joan DeRoy
Louise Newell
Martha Poulos
Hildegarde Rolander
Joan Griswold
Elaine Hamilton
Margaret Hubbuch
Barbara M. Kaiser
Barbara Knowles
Jeanne Morin
Susanne Seely

Children's Hospital Society

Elmeene B. Chain
Clyde R. Holway
Thomas B. Irwin
Marion E. Mankin
Adeline Rodrigues
Mary Alice Hill
Grace Suchiko Ishida
Barbara Schamp
Mark Crutcher, Jr.

D. T. Watson School of Physical Therapy

Mary L. Agresti
Helen L. Bauman
George D. Brunner
Elizabeth A. Buschow
Shirley I. Campbell
Antoinette Carpenter
George E. Cotton
Mary J. Devlin
Dorothy M. Dizneff
Jean B. Dudek
Nancy L. Fix
Ruth S. Hayden
Ann E. Hesch
Dorothy E. Kasputys
Florence R. Levendosky
Selvin T. Lewis
Mary Ann Liebermann
Carolyn A. Lindsey
Margery Lynch
William Menko
Jean A. Martens
Mary G. Martz
Robert G. Maul
Jack W. Miller
Virginia W. Newton
Orfa N. Pagan
Clifford E. Porterfield
Fatima F. Rieger
Hazel E. Robertson
William W. Simon
Henrietta Smith
Shirley E. Smith
Dallas Sterling
Ralph E. Weaver
Edith E. Wilson
Raymond J. Wojciak

Mayo Clinic

Eleanor Hammond
Sarah Anne Smith
Bernard Kimmel

New York University School of Education

Lemel Dov Aldubi
George Andrews
Edwin B. Augustin
Norton Baskin
Rudolph L. Bloch
Eleanor Bonn
Robert H. Christensen
Oscar Cohen
James M. Cox
Henry DiRe
Richard Freeburg
Walter Givens
Richard M. Herman
George Kamilar
Leonard L. Lasky
Martin Levy
Virginia Marin
Ellie D. McDew
John T. McMahon
Harold E. Potts
George A. Rickert
Stanley Rosenblatt
Otto Strobino
James Weaver
Margaret Wellington
Robert Zebner
John Badenhop
Frank Connolly
Harry Elazarian
Albert K. Fairbanks
Milton Fried
Abraham Katzowitz
Joseph Marino
Robert D. Mears
Frederick Murko
Mercedes Randall
Alvin Rogerson
John D. Galloway
Gerald Weiss
Katrine E. White
Jeremiah Wyatt

University of California

Thelma Chinn
George E. Cousens
John R. Eaton
Mrs. Geraldine Gorman
Daniel P. Halstead, Jr.
Elna M. Hauser
Otto Hirsch
Lorraine Kovach
Joseph E. Mulroy
Niels P. Murer
Garland M. Smith
Mrs. Joyce W. Stevens
Ermine Duccini
Mrs. Marisa Lusiardo
Robert Nochta
Marion T. Oishi

University of Minnesota

Oliver Thomas Bogro
Glenn H. Bostrom
Donald Earl Erickson
Gertrude A. Freeman
D. LaVonne Jaeger
Mrs. Donna M. McArdle
Mrs. Idell D. Moulton
Constantine Valentine
Robert O. Wood

Office of Vocational Rehabilitation

Mary Switzer has been appointed as Director of the Office of Vocational Rehabilitation, succeeding Michael Shortley. Miss Switzer has been assistant to the administrator of the Federal Security Agency since 1939, with coordination of the Agency's public health and medical interests her chief responsibility.

Abstracts

Care of the After-effects of Poliomyelitis: Forearm and Hand

Louis P. Britt, M.D., and Robert L. Bennett, M.D., Warm Springs. In ARCHIVES OF PHYSICAL MEDICINE, 31:10:646, October 1950.

When it is realized that next to the brain the hand is the greatest asset of man, the importance of achieving a maximum functional result is apparent. The ultimate functional capacity depends upon the effectiveness of the early and convalescent care of the surviving motor units. This treatment obviously begins as soon as the diagnosis is made and the patient's general medical condition permits.

Early care of the involved segment begins the moment the patient's condition permits and should embrace three major objectives: (1) relief of pain, (2) release of tightness, (3) support of weakness. Relief of pain may be accomplished by local application of heat in the form of warm, moist fomentations and support in the position of comfort. It should be noted that any position which affords comfort is permissible in the very early stages, and transition to the functional position should be made under the dictates of pain. With relief of pain, the release of tightness may be furthered by passive motion to achieve gradually a painless, functional range of motion in each joint.

During the early phase, the evaluation of weakness should be based upon observation of movements and gentle palpation of tensions in muscle groups. As soon as a painless, functional range of motion is obtained, a detailed muscle test should be made. During this phase of treatment, the early convalescent period, the individual muscle or muscle group is the unit specifically involved needing muscle reeducation. Early detection of weakness in the long finger extensors, intrinsics of thumb, lumbricales, and first dorsal interosseus is especially important, since prognosis for recovery is poor if treatment is delayed.

Coordination is the keynote in muscle reeducation, and all motion (passive, active-passive, active, and active-resistive) must be carried out in the normal pattern. Strengthening of the individual muscles or muscle groups should be stressed only after coordination has been firmly established. During the early phase of muscle reeducation, certain supportive and/or assistive apparatus may be necessary for fulfillment of the normal pattern of motion.

The question of when to discontinue the painstaking muscle reeducation and concentrate on increasing the functional capacity of the forearm and hand by any means available is an important one. Certainly, coordination must be established and the peak of individual muscle strength obtained. A plateau in the periodic muscle tests will readily reveal that maximum muscle strength (compatible with central nervous system involvement) has been reached in this treatment regimen.

A complete evaluation of the functional capacity must be determined before a specific program of functional training can be instituted.

A discussion of surgical procedure is beyond the scope of this paper. It should be remembered, however, that the surgical results are greatly influenced by the caliber of early conservative care. Prevention or minimization of deformities and attainment of maximum coordination and strength will make surgery easier to perform and will facilitate the functional capacity. Surgery should not be considered the final step in the treatment program. Instead, it should be considered another step in the over-all program; its timing should be dictated by the necessity of increasing functional activities and not to any specific chronological phase of the treatment.

Educational Therapy in the Rehabilitation of the Tuberculous

Edward F. Gordon, M.D., and Estelle C. Phillips, M.A., Staten Island, N. Y. In ARCHIVES OF PHYSICAL MEDICINE, 31:9:595, September 1950.

All the objectives of educational therapy which have been discussed are obviously interrelated in almost every case. It is merely for the sake of clarity that certain groupings have been selected to illustrate the significance and efficacy of this modality. For example, the "hardening process" is inseparable from provocational exploration or avocational pursuits. The establishment of new work habits may be related to the psychotherapeutic effects of all work activity. Any one or all of these factors may have to be considered in relation to the total personality pattern of the patient during the administration of the broad rehabilitation aspect of purposeful educational therapy. This interrelationship is borne out by the assertion of a well-known educator and philosopher, that the fully adjusted and well-rounded individual has four facets to his personality to be developed—vocation, avocation, family group membership, and community group membership.

At whatever educational level or with whatever complexity of purpose work activity be administered, the educational therapy unit, as an integral part of the rehabilitation program, provides a therapeutic means of aiding in the prevention of relapse. It assists the patient to restore himself to that degree of complete personal and economic security which will be within the limits of his physical capacity. It enables him to attain through achievement that self-confidence which makes for an active and useful life. In short, to paraphrase Plato, his disease may "prove an unexpected benefit" if the lesion in his lung compels him to reorganize his life.

The Mechanics of Deformities of the Hands in Atrophic Arthritis, and a Discussion of Their Prevention and Correction

James C. Small, M.D., F.A.C.P., Philadelphia. In ANNALS OF INTERNAL MEDICINE, 32:6:1087, June 1950.

One of the earliest deformities in atrophic arthritis occurs in the hands. Characteristically, it develops into an ulnar deviation of the fingers with contractures at the metacarpophalangeal joints. This so greatly reduces the ability to use the hands that it may be regarded as one of the most distressing deformities of the disease. The morale of patients usually suffers less from their inability to walk than it does from inability to use the hands in some time-consuming occupation which might tend to shorten the long waking hours. Since more than 90 per cent of patients with this disease are women, this unsightly deformity wounds the patient's pride more than any other aspect of the disease. It cannot be hidden and constantly serves to advertise the affliction.

An anatomic imbalance in the antagonistic muscles which flex and extend the fingers is presented as the primary cause of the ulnar deviation deformity of the hands in atrophic arthritis.

A simple therapeutic exercise to strengthen the extensor muscles is suggested to correct and prevent these deformities.

The Concept of "Acceptance" in Physical Rehabilitation

Morris Grayson, M.D., New York City. In THE MILITARY SURGEON, 107:3:221, September 1950.

"Acceptance" is a term variously applied and of essential significance in rehabilitation of the physically disabled. The term is loosely used in rehabilitation circles and is usually understood by implication rather than by definition. Webster defines acceptance as "the act of receiving (a thing offered) with a consenting mind," or "the act of receiving or admitting and agreeing to." By its very definition, it implies an active and very individual process of "agreeing to" or having a "consenting mind." Translated into symptomatology commonly met with in physical rehabilitation, the term is usually applied to three aspects of the individual's rehabilitation—physical, social, and psychological. Physically, it implies that the patient is well aware of the nature of the disability, its origins, its complications, and its prognosis. Socially, it implies that the patient is realistic toward his job, toward housing, family relations, etc. Psychologically, it implies that the patient is showing no serious emotional symptoms referable to his disability.

Acceptance is the psychological key to physical rehabilitation.

The psychodynamics of acceptance involves two processes, (a) psychobiological incorporation (the body image) and (b) social integration (the reality principle).

Acceptance and rehabilitation can take place without a physician's aid, but in many cases, a center of rehabilitation is necessary.

A psychiatric team functions by direct psychotherapy to the patient and indirectly by education of other workers in the rehabilitation team to the individual psychological needs of the patient.

Clinical Management of Acute Poliomyelitis

Emil Smith, M.D.; David J. Graubard, M.D.; Joseph Falcone, M.D.; Thurman B. Given, M.D.; Philip Rosenblatt, M.D.; and Avner Feldman, B.S.; Brooklyn. In THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:3:213, September 16, 1950.

At the present time there is no form of treatment in acute poliomyelitis that limits the spread of virus in the central nervous system or alters its effect on the nerve cells. Many patients recover completely without treatment, but others, despite heroic measures, suffer extensive paralyses.

In the past few years there has been an increasing interest in the control of pain and vasomotor instability. Various means of sympatholysis have been tried to correct the vasomotor dysfunctions. Maintenance of muscle circulation aids in its rehabilitation, and the degree of recovery is directly related to the time of institution of corrective measures.

Our objective has been to shorten the duration of pain, muscle spasm, and vasomotor instability during the acute and subacute stages of poliomyelitis, so that measures for rehabilitation of the patient can begin as early as possible. It is our belief that priscoline has been a useful adjuvant in the early management of the disease. It has allowed us to eliminate use of moist hot-packs and has enabled us to handle a large number of patients in this past epidemic with more comfort and without taxing our limited nursing staff. Patients could be transferred for orthopedic management earlier than in previous years. At the very least the further investigation of priscoline and of other sympatholytic drugs is indicated.

Summary. (1) During the year 1949, 665 patients with acute poliomyelitis were treated with priscoline (2-benzyl-4, 5-imidazoline) hydrochloride, a sympatholytic drug.

(2) The benefits to treated patients included the relief of pain, spasm, and vasomotor dysfunction.

(3) In the dosage plans described, morbidity was minimal.

(4) Further investigation of the use of sympatholytic drugs in acute poliomyelitis is indicated.

A Discussion on Muscular Contraction and Relaxation: Their Physical and Chemical Basis

A. V. Hill, F.R.S., London. In PROCEEDINGS OF THE ROYAL SOCIETY, Series B, 137:886:40, April 13, 1950.

The elementary unit of contraction in skeletal muscle is a single twitch, evoked either by a single nerve impulse arriving at an end-plate or by a direct electric shock. It is a very rapid affair, requiring for completion a time varying from a few milliseconds to a fraction of a minute according to the muscle and its temperature. It is followed by a slow recovery ("recharging") process taking from a few minutes to an hour.

The focus of the present discussion is the relation between (a) known mechanical, thermal, electrical, and other physical changes accompanying contraction and relaxation and (b) the underlying chemical causes.

Pain and Contracture in Poliomyelitis

Arthur C. Guyton, M.D., and Robert C. Reeder, B.A., University, Miss. In ARCHIVES OF NEUROLOGY AND PSYCHIATRY, 63:6:954, June 1950.

On the basis of personal experience of one of us (A.C.G.) and of his contacts with other patients, the pain of poliomyelitis is described as that commonly observed after a muscle bruise or after severe exercise, or that of a stiff neck after one has been sleeping in a cold breeze.

Experimental studies on ischemic pain in the arm of human subjects suggest that this type of pain is not the same as that of poliomyelitis.

Studies on ischemia in the legs of dogs, as well as in the arms of human subjects, showed that blood flow must be stopped 90 to 95 percent of the time in order that pain may be produced. There is no evidence that this degree of ischemia exists in the muscles in poliomyelitis.

In twenty-five dogs, anterior rhizotomy, a type of lesion physiologically resembling the lesion of poliomyelitis, invariably caused extreme sensitivity in the affected leg. Contracture, typical of that seen in poliomyelitis, also followed. It is concluded, therefore, that the pain and contracture in poliomyelitis are probably due to local pathologic changes in the muscle following denervation.

It is postulated that the pain of poliomyelitis may be caused by products of cellular degeneration and that the value of vasodilatation in the treatment of this symptom is the increased removal of such products.

Neurogenic spasm, as a cause of contracture and pain, is discussed, and it is concluded that there is no evidence to support such a concept, chiefly because even totally paralyzed muscles undergo the same type of contracture as do other muscles.

Muscle Spasm and Paralysis in the Diagnosis of Acute Poliomyelitis

George J. Boines, M.D. In MEDICAL RECORD, 163:8:204, August 1950.

The procedure we have found most valuable for diagnosis of acute poliomyelitis in the early or febrile stage (*i.e.*, in the first five to seven days), and by which a high percentage of wrong diagnoses may be avoided, is to follow the symptoms and signs presented by the muscle condition as originally described by Pohl. The teaching that, because the virus attacks the anterior horn cells, flaccid paralysis has to be present before the diagnosis can be made, has not been borne out by our experience. Practically every acute poliomyelitis patient presents muscle spasm and every paralytic patient presents incoordination, weakness, and paralysis of the muscles in addition to a severe degree of muscle spasm.

It is evident that the affected muscles must be isolated and rated as soon as the acute spasm is over so that the exact involvement of the patient's muscles may be known. It is also highly important for the physician to instruct the technician with regard to the muscles or muscle groups which require stretching reeducation and coordination, so that weak muscles may be strengthened, and, thus, deformity of the involved skeletal parts avoided.

It is our experience that such close observation of the symptoms and signs presented by muscle condition as we have indicated here permits a diagnosis of poliomyelitis in over 95 percent of the cases in the first five to seven days of the disease.

Physiological and Performance Changes in Athletic Conditioning

F. M. Henry and W. E. Berg, Berkeley, Calif. In JOURNAL OF APPLIED PHYSIOLOGY, 3:2:103, August 1950.

During the recent war period there was widespread use of performance tests for the assessment of "physical fitness." In general, these tests took the form of standardized runs and gymnastic stunts, the former presumably measuring speed or endurance, depending on the length of the run, while the latter tested agility. Fitness for muscular work was also measured by step tests and pack tests, which were thought to be improvements over running tests because they were better standardized and less influenced by special skills. *A priori* validation of these procedures, while perhaps justifiable as a temporary expedient, is not acceptable as a true measure of their value.

Determinations of oxygen debt and carbon dioxide surplus during the pay-off period after a moderate standard exercise were made on athletes before and after a regimen of physical conditioning. Performance scores were also secured on a 300-yard run, shorter runs, and stool-stepping to exhaustion.

All measures improved in the theoretically expected direction. Oxygen debt and carbon dioxide production were more effective than performance tests as measures of improvement in physical condition. Conditioning produced a more significant change in amount of debt than in the rate of pay-off. Among the performance tests, the longest run was most effective and the pack test least effective for measuring improved condition. The RQ of the recovery metabolism was not significantly affected by conditioning; neither was there an effect on the ratio index of endurance in running. Individual reduction in oxygen debt due to conditioning tended to show a small positive correlation with individual improvement in performance. In general, there was no correlation between size of the individual oxygen debt and individual performance scores. The experimental results were explained by a theory that individual performances in exhaustive exercise are largely determined by psychological factors and specific skills, rather than ultimate physiological limits. These tend to balance out in subtractive improvement scores, so that increased capacity can be demonstrated as a result of athletic conditioning. Actual fundamental capacity can be determined only by more direct methods such as oxygen debt measurements.

Rehabilitation of the Tuberculous Patient in the Veterans Administration

Louis B. Newman, M.E., M.D., Chief, Physical Medicine Rehabilitation Service, Veterans Administration Hospital, Hines, Ill. Read at the First Annual Tuberculosis Rehabilitation Conference sponsored by the Tuberculosis Institute of Chicago and Cook County, Chicago, Ill., Feb. 23, 1950.

Although tuberculosis is a communicable disease which can be prevented, the problems associated with the rehabilitation of tuberculous patients are many. However, a proper functioning, well-integrated program of total rehabilitation will be an important factor toward the possible solution of these problems.

As with other disabilities, we must not only be concerned with the physical progress and well-being of the patient, but also with his mental, social, psychologic, and vocational aspects. Medical feasibility for training and/or employment must be carefully considered and evaluated.

The Physical Medicine Rehabilitation Service dealing with the care of tuberculous patients consists of physical therapy, occupational therapy, educational therapy, and manual arts therapy. In order to further integrate the activities of the services at the hospital, a Tuberculosis Rehabilitation Board has been constituted.

The objectives of physical medicine rehabilitation in the Veterans Administration are as follows:

1. To promote acceptance of bed rest by the patient by providing relaxation therapy when indicated.
2. To furnish facilities for developing and measuring work capacity.
3. To provide pre- and postoperative therapy for selected patients who require thoracic surgery.
4. To help motivate patients to develop an active interest in, and a desire for, complete recovery.

Work capacity is the amount of work the individual can do safely and without adversely affecting his physical or mental state of health.

It is the job of those concerned with rehabilitation to motivate the patient, as a properly motivated patient is a cooperative one. It is well recognized that as a rule the most can be accomplished for the patient who cooperates. Further, it is important that the patient have a complete understanding of what is to be done, how it is to be done, and the importance of his willingness to accept the care and guidance of those in the field of rehabilitation. An educational program is also essential for the patient's family and the public in general.

Role of Physical Therapy

The patient is usually referred to the physical therapy section sometime within the first week following his admission to the Tuberculosis Service. He is given instruction in scientific relaxation, including correct bed posture, pillow arrangements, and diaphragmatic breathing.

In those patients requiring thoracic surgery, such as thoracoplasty, physical therapy is prescribed in order to secure the following:

1. Correct posture, which will include bed posture training, as well as correct posture for all activities, including sitting, standing, and walking.
2. Maximum range of motion and function of the upper extremity on the operated side.
3. Maintenance of proper mobility of the neck, shoulders, and spine.
4. Maintenance of muscle tone and strength of the entire body by controlled, specific exercises.

With adequate medical care and the combined teamwork of all engaged in total rehabilitation, together with proper follow-up for a sufficient time, these individuals with tuberculosis can return to a productive life, with a full measure of happiness, contentment, and satisfaction.

A continued concerted drive to enlighten the public on all aspects of tuberculosis such as the etiology, prevention, treatment, etc. is most essential. Further, all organizations and agencies, governmental and otherwise, associated with the care of the tuberculous patient can, by frequent exchange of ideas and careful analysis of the existing problems, be in better position to solve them.

Our reward will be in the prevention of tuberculosis. However, once the individual is infected, then our hopes will lie in early recognition of the disease together with prompt, effectual treatment and follow-up. *Rehabilitation must be total and dynamic.*

Care of Children Convalescing from Poliomyelitis: Discussion of Several Points of Controversy

Robert L. Bennett, M.D., Warm Springs. In JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:5:377, September 30, 1950.

After the period of virus invasion is over and progressive damage to the central nervous system threatening the child's life is past, the physician is faced with the problem of intelligently handling his young patient while the inflammation in the neuromuscular system quiets down and the remaining anatomically intact neuromuscular units become physiologically available for use. He must be responsible for a logical progression of treatment procedures that will, first, restore the most effective use of all remaining neuromuscular units, and, second, minimize all musculoskeletal deformities that might limit the child's ultimate functional capacity. Even in the mildly involved patient (infant or child) this means years of care while the individual increases in height, weight, activity, and responsibility. During every day of these years there is the constant threat of progressive weakness through misuse of muscle action and progressive skeletal deformity through excessive or unprotected activity.

The over-all program is similar in all responsible and experienced medical units caring for patients with poliomyelitis and its after-effects. If the programs are so uniform, why does there exist so much controversy and indecision within each step, and why are end results often so inexcusably poor? The major points of conflict can be narrowed down to four issues: (1) hospitalization, (2) physical therapy, (3) inactivity, activity, and apparatus, and (4) surgery.

The period of hospitalization in poliomyelitis will depend on three factors, the amount of involvement, the age of the child, and home conditions. A convalescent hospital unit should be used only for the period of time necessary (1) to evaluate accurately the child's involvement, (2) to set into action the basic routines of mobilization and strengthening, (3) to order, fit, and begin the use of necessary assistive and supportive apparatus, and (4) to instruct the parents thoroughly in home routines of care. The child may be expected to require years of treatment. The great bulk of this treatment must be carried out at home by the child and his parents. Only in severe and unusual problems is there need to hospitalize the child more than three to six months after onset of illness.

All too frequently the treatment program keeps the child hospitalized after the acute stage for six, twelve, or even eighteen months and then the physician spends fifteen minutes presumably checking the child every three to six months in a crippled children's clinic until the patient is old enough for surgical treatment. It is far better to hospitalize the child for a shorter period initially and to readmit him at regular intervals for short periods of evaluation, specific treatment, fitting and instruction in use of apparatus, and reinstruction of parents. It is only when faulty or inadequate care is given that long periods of hospitalization are necessary.

There is little doubt that physical therapy should be used and is the method of choice in convalescence after poliomyelitis, but there is much indecision about what type should be used, when it should be started, and how long it should be continued. The tools should not be criticized for their inadequacy in the hands of poorly trained and unskilled physicians and physical therapists.

In the early phases of convalescence, physical therapy has the great responsibility of helping to relieve pain and to restore and maintain normal mobility of the involved bodily segments. Later, through specific forms of exercise based on fundamental principles of functional anatomy and muscle reeducation, physical therapy assists the patient to use the muscle fibers remaining under his voluntary control with such coordination that, with guided use, effective strength and function are regained.

As the need for certain orthopedic apparatus becomes apparent, physical therapy adapts its routines to include the most efficient and beneficial use of this apparatus. Much indecision and controversy exists as to how long one should continue attempts to coax back strength and function in severely weakened muscles. Much time and money can be wasted by prolonging this phase of muscle reeducation long past the period when any reasonable hope for the return of strength has vanished. On the other hand, muscle reeducation terminated before possible voluntary control has returned and coordinate patterns of movement are set is an equal waste. Here one may be guided, but not ruled, by the usual relationship of recovery of individual muscle strength as related to time.

If it becomes obvious that severe functional handicap will always be present, physical therapy then attempts to instruct the patient in the use of special apparatus to overcome specific functional problems. This requires great ingenuity and patience, and the apparatus must be changed as the child grows and his functional demands change.

The next points of controversy may be grouped together because of their close relationship: inactivity, activity, and the use of apparatus. It may be said that the future of the child with even the mildest after-effects of poliomyelitis lies in the delicate balance of activity against inactivity. Relative inactivity, with the

proper support of weakened bodily segments, is necessary to protect denervated muscle fibers and maintain normal skeletal alinement. Some activity is not only desirable but inevitable in the infant or child, who soon manifests a normal and healthy desire to move about despite muscle weakness.

Musculoskeletal deformities develop from one cause: persistent faulty use of muscle strength and/or persistent faulty skeletal alinement. Equally severe problems arise with faulty bodily mechanics of the patient at rest as during activity. The proper use of orthopedic apparatus for support, assistance, and, if necessary, correction is the only reasonable and practical answer.

Activity, such as turning over in bed, sitting up in bed or in wheelchair, and standing and walking, should be started as early as it can be done safely.

In the early stages of convalescence, apparatus must be thought of as a protection permitting safe activity while the basic program of rest and specific muscle reeducation is being carried out. Apparatus may also be used to prevent, minimize, or correct deformity. Later, when severe loss of muscle strength is evident, apparatus is used to support and assist activity and to minimize the danger of progressive deformity as the child increases in height, weight, and activity.

Certain orthopedic surgical procedures may be indicated during the months and years that are commonly considered to be within the period of convalescence. The necessity for these procedures usually arises from the need to reestablish adequate mobility of bodily segments by release of fascial or ligamentous structures when the more conservative methods of manual and mechanical stretching have failed.

Pain and Contracture in Poliomyelitis

A. C. Guyton and R. C. Reeder. In ARCHIVES OF NEUROLOGY AND PSYCHIATRY, 63:843, June 1950. Abstracted in JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:4:339, September 23, 1950.

Guyton and Reeder observe that the patients with acute poliomyelitis is often much more concerned with the pain than he is with the paralysis. Patients have been known to remain awake for an entire week as a result of the pain, yet little is known of its cause. One of the authors, Guyton, had had a severe attack of poliomyelitis in 1946. His personal experience and what he learned from patients convinced him that the pain of poliomyelitis is like that commonly observed after a muscle bruise or after severe exercise, or like that of a stiff neck after one has been sleeping in a cold breeze.

The authors describe experiments carried out in order to learn more about the pain of poliomyelitis. Ischemia was induced in dogs and in human subjects. The anterior roots of the spinal cord in dogs were cut, with preservation of the posterior roots from the same segments.

Ischemic pain in the arm of human subjects is not the same as that of poliomyelitis. Anterior rhizotomy in twenty-five dogs, a type of lesion physiologically resembling the lesion of poliomyelitis, invariably caused extreme sensitivity in the affected leg. Contracture, typical of that seen in poliomyelitis, also followed.

It is concluded, therefore, that the pain and contracture in poliomyelitis are probably due to local pathologic changes in the muscle following denervation. The pain of poliomyelitis may be caused by products of cellular degeneration. Vasodilation may be valuable in the treatment of this symptom, because of the increased removal of such products. The authors discuss neurogenic spasm as a cause of contracture and pain and conclude that there is no evidence to support such a concept, chiefly because even totally paralyzed muscles undergo the same type of contracture as do other muscles.

Mechanism of Suffocation in Spinobulbar Poliomyelitis and Experiences with Operative Treatment

Arne Sjoberg, M.D., Stockholm, Sweden. In ARCHIVES OF OTOLARYNGOLOGY, 52:3:323, September 1950.

During the last five years a wave of enthusiasm for tracheotomy in bulbar poliomyelitis has swept over the United States. At the First International Poliomyelitis Conference, held in New York from July 12 to 17, 1948, it was generally agreed that in certain cases tracheotomy must undoubtedly be considered as directly lifesaving.

Preliminary Conclusions

1. The majority of fatal cases in the early stages of infantile paralysis are, most probably, in accordance with the American opinion, primarily due to suffocation—anoxia—and in all probability not to circulatory paresis.

2. According to Bergman and Huldt, the mortality rate for patients treated with the respirator during the twelve-year period (1934 through 1945) is 86 percent (119 survived of 854) for the whole of Sweden and 82 percent (20 of 158 survived) for Stockholm.

3. So far, thanks to the operative treatment, 12 or 14 patients, whose condition has been considered hopeless up to now, have been saved.

4. The indications for operative treatment coincide with the immediate cause of suffocation and can be divided into the following five groups.

(a) Peripheral respiratory paresis with secretional retention.

(b) Aspiration (of saliva and vomitus) in purely bulbar cranial nerve lesions (above all, in paralysis of the vagus).

(c) Bilateral paralysis of the laryngeal abductors combined with two-sided paresis of the pharynx.

(d) Spinobulbar lesions, manifesting themselves, among other ways, in a bilateral paresis of the pharynx (plus, in certain cases, two-sided nuclear paresis of the accessory nerve, plus injuries to the first and second cervical segments of the spinal cord). The suffocation picture in these cases is dominated by the angle, or rope dent, symptom, which constitutes an alarm signal for the nursing staff. The patient runs the risk of being hanged or strangled any minute by a contracture or tonus excess in his nonparalyzed antagonistic muscles, which draw the hyoid bone backward and upward. If, in addition, a nuclear paresis of the accessory nerve sets in, the rope dent symptom is further accentuated and the patient's condition is aggravated, owing to a contracture of the non-paralyzed muscles which bend the head backward.

(e) Cases in which the operative treatment is applied early, prophylactically.

In all types of respiratory paresis, the respirator treatment may be directly harmful and can even hasten death by suffocation if bronchoscopic suction with or (in exceptional cases) without tracheotomy is not applied simultaneously.

Postural drainage with oxygen or carbogen is absolutely necessary. During the first, and sometimes even during the second, week the fluid balance must be carefully controlled and the liquid administered only parenterally in isotonic or hypertonic form. Dextran and blood transfusions have proved to have a beneficial effect on the initial shock condition, which often appears in the hours following a tracheotomy.

Osteoarthritis and Body Mechanics

F. X. Krynicki, M.D., Detroit. In THE JOURNAL OF THE MICHIGAN STATE MEDICAL SOCIETY, 49:9:1070, September 1950.

Most authorities speak of osteoarthritis occurring in persons past forty but not commonly before that age. In the absence of rheumatoid arthritis, of rheumatic fever, or of any of the usually recognized chronic arthritides in an individual under forty, a void is left in the diagnosis of chronic arthritis. Faulty body mechanics suggests an answer because much of the pain can be eliminated by the correction of such faults.

The correction of faulty body mechanics in arthritis and in the rheumatic diseases has been advocated by authorities for a long time.

Treatment.—When a patient wears the special type of shoe recommended, his foot posture tends to approach the ideal, and the various joints tend to become realigned. Uncomplicated neuralgias tend to disappear in a relatively short time. Moreover, when irritation of or about the joint is not too great, the mere correction of foot posture is sufficient to eliminate the painful symptoms. If, however, there is a great deal of irritation from torsion or unequal joint pressure resulting in constant or severe pain, treatment for their alleviation must be added. It is here where the present-day standard therapy of osteoarthritis is of value, which consists, in a word, in the use of salicylates, analgesics, parenteral foreign protein (vaccines, sulfur, and so on), intravenous procaine, physical therapy, local procaine injections, and so forth.

Exercises are held to a minimum. Many patients with osteoarthritis, with or without backache, are exhausted, and on arising in the morning feel as though they had not slept at all. It did not seem proper to request exercises under these circumstances. Moreover, some of the standard exercises require day-long concentration, which is hardly possible except under direct and constant supervision such as may be obtained in a clinic.

Summary

(1) Confirmation is presented of Laurence Jones' work on low backache and various neuralgias.

(2) It has been shown that many of the symptoms associated with arthritis are measurably due to faulty body mechanics, and

(3) That these symptoms are reversible by correction of faulty body mechanics, and

(4) That this in turn is accomplished by correction of faulty foot posture.

(5) Ideal foot posture is maintained by wearing specially prepared supportive shoes.

(6) In the general treatment of osteoarthritis and fibrositis, the combination of routine medical and physical therapy, for irritative changes, with correction of faulty foot posture, leads to a symptomatic response superior to that which is ordinarily obtained.

Rehabilitation of Patients with Hemiplegia

I. F. Nash, M.D., St. Pauls, N. C. In SOUTHERN MEDICINE AND SURGERY, 112:9:295, September 1950.

Little of a helpful nature in the management of cases of hemiplegia was taught any of us in college or hospital. An outline as promising of results as that given by Yamshon will find general welcome, to the profit of many a patient.

In cases in which paralysis has been caused by cerebral hemorrhage, activity must wait until all evidence of bleeding ceases. Patients who have had cerebral embolism or thrombosis can be started on treatment within forty-eight to ninety-six hours. Initially, atonicity is usually present. This pseudoflaccid stage later changes to a spastic stage. The muscles on the involved side begin to atrophy immediately as the result of disuse. The involved muscles will respond, by contraction, to electrical stimulation. Electrical stimulation can delay atrophy even in muscles in which peripheral nerves have been severed. To minimize atrophy of disuse, the paretic muscles are electrically stimulated. In the later stages when hypertonicity appears, and with it some degree of voluntary motion, electrical stimulation is no longer indicated.

During the atonic phase, it is important to maintain the range of motion in the joints. Contractures are liable to develop, particularly in older individuals. The commonest site of contracture is the shoulder. Contracture of the ankle in plantar flexion, while common, is more apparent than real. At this stage, mobility is maintained by passively moving each joint through its complete range of motion at least once a day.

Occupational therapy procedures, which have the advantage of keeping the patient interested in improvement, are best suited to treatment of the arm. Functional treatment by occupational exercise should be started with the arm at shoulder level. The arm may be supported either on a table or by a sling suspension. Resistance is added as tolerated. With increase in strength, the support may be lowered so that work is done at a lower level, and ultimately the support may be removed. Then working position of the unsupported arm may be gradually raised so that it has to be held against gravity.

Respiratory Studies in Paralytic Poliomyelitis

R. B. Bourdillon, C.B.E., D.M.; E. Davies-Jones, M.B., B.S., D.A.; F. D. Stott, D.Ph.; and L. M. Taylor, B.M., B.Ch., Oxford. In BRITISH MEDICAL JOURNAL, No. 4678, September 2, 1950.

The following notes are based on studies at the Western Hospital, Fulham, by two of us (L.M.T. and E.D.-J.) during the 1949 epidemic of poliomyelitis, and on laboratory studies at Stoke Mandeville Hospital. While many of the conclusions are tentative, it has become clear that there is much need for improvement in both the design and use of breathing machines. These notes are published in the hope of facilitating such improvement.

Numerous measurements of tidal air have been made with a spirometer on patients and on healthy subjects in Both and Drinker respirators, and on patients after respirator treatment for poliomyelitis. These measurements show a wide range of variation from day to day in most cases.

It is considered that in the treatment of poliomyelitis with respiratory involvement there is much need for measurements of vital capacity and tidal air, unless frequent measurements of blood O_2 and CO_2 are practicable.

Early measurements of vital capacity are suggested in order to detect the first signs of respiratory involvement, with a view to resting the muscles concerned by the prompt use of a respirator. The tidal air measurements are indicated as a rough guide to the correct setting of respirator pressures so as to avoid under- or over-ventilation.

The potential dangers of hyperventilation are discussed, and measurements are given showing the ease with which this is produced in respirators.

Tests on the use of the prone position in respirators are described. The importance of using this position is emphasized, especially in bulbar cases.

Some possible improvements in the Both respirator are discussed, in particular, provision for slower speeds and a reduction in the angle of slope and in the thickness of the front panel.

Poliomyelitis of Newborn: Pathologic Changes in Two Cases

I. L. Baskin, E. H. Soule, and S. D. Mills. In AMERICAN JOURNAL OF DISEASES OF CHILDREN, 80:1, July 1950. Abstracted in JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:7:575, October 14, 1950.

Baskin and co-workers report two infants with acute poliomyelitis in the first two weeks of life. The first infant, an active normal boy, was born while his mother was in a respirator, critically ill with poliomyelitis of four days' duration. The infant was isolated immediately on delivery. The baby's course was uneventful for three days, when a rectal temperature of 101° F. developed. Listlessness and cyanosis of the lips and nail beds were noted two days later. Response to pain was absent in the right foot and leg and minimal in the left. The patient became rapidly worse, with extreme flaccidity, irregular respiration, and progressive cyanosis. Death occurred on the seventh day of life.

In view of the fairly well-established fact that the minimal incubation period of clinical poliomyelitis is five days and that immediate isolation of the infant was carried out on delivery, his illness either appeared after an incubation of only three and one-half days or resulted from an intra-uterine infection. The second infant was a normal boy whose mother had acute poliomyelitis on the second day after the delivery, while the baby showed no signs of illness. Rectal temperature of 101° F. and listlessness were noted on the eighth day of life. The next day the extremities became flaccid and respirations irregular and rapid. Death occurred on the fourteenth day of life. Necropsy revealed that the significantly involved parts of the central nervous system were confined to the cord and the medulla. On the basis of the evidence presented, it is no longer possible to assume that newborn infants are immune to poliomyelitis.

Intermittent Treatment of Poliomyelitis with Progressive Resistance Exercise

Sedgwick Mead, M.D., St. Louis. In JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:6:458, October 7, 1950.

The first report on the use of progressive resistance exercise in poliomyelitis is that of De Lorme, Schwab, and Watkins in 1948. Every report since that time has been an enthusiastic confirmation of the value of this form of treatment, without a single dissent. As a result of my experience with the method since the early part of 1947, I believe that it is by far the most important contribution yet made to the treatment of this disease.

There has been sometimes justifiable criticism of the use of physical therapy in poliomyelitis and cerebral palsy. This criticism must be met by increasing use of quantitative measures of progress such as are provided by the technic of progressive resistance exercise. The manual muscle grades (Lovett) are inexact but must also be used until better measures are devised. Goniometric measurements are simple and obvious. It is the duty of the occupational therapist to test the patient against a checklist of daily living activities. She can also help the patient to recover coordination and skill.

Quantitative determinations not only reveal progress but show when it is lacking. When three or four determinations of the resistance maximum all give the same result, even the patient gets the point and is willing to discontinue treatment. The patient with an absolute limitation is thus given realistic insight into his disability and encouraged to learn how to live within it. Without such quantitative data it is possible for the unscrupulous or self-deluded physician to continue treatment of the patient for preposterous lengths of time, holding out vague promises of future cure.

It has been asserted that physical therapy in poliomyelitis is a useless luxury because the patient, by the physiologic demands of his home activities, will recover to the fullest possible extent without additional exercise. The final case report is pertinent to this objection.

The rehabilitation of the patient as a whole is presumed. Stabilizations, transplants, epiphyseal arrests, vocational testing, and training are as important as they ever were.

Summary

1. The use of progressive resistance exercise in convalescent poliomyelitis is described.

2. A few weeks of intensive daily exercise is alternated with three or four months of home activities until no further improvement occurs.

3. Advantages of this regimen over continuous treatment include economy of effort and means, more efficient use of equipment and personnel and better psychological adjustment. Most important is the quantitative measure of progress provided.

An Approach to Dynamic Posture Based on Primitive Motion Patterns

Julia Singer Heller, M.D., Arno David Gurewitsch, M.D., New York. In ARCHIVES OF PHYSICAL MEDICINE, 31:10:632, October 1950.

The concept of posture has undergone frequent changes during the last decades. Until recently good posture was defined as a static position of standing such as seen in military schools. This position is based on conscious active contraction of different muscle groups. It cannot be held for any length of time, and it is uneconomical, owing to contraction of many muscle groups which normally would not be called into action, or at least not to the same extent. This position also impedes normal breathing through the active contraction of the upper abdomen, and it is artificially superimposed on the natural play of postural reflexes in the normal standing position.

The present trend deviates more and more from this static concept of posture toward a more dynamic one, taking into account the different postures occurring in daily life.

Correct dynamic posture is an outcome of the physiological use of the body as an entity.

If we attempt to analyze dynamic posture, we can define three main components: muscle balance, grading of muscle contractions, and timing of muscle contraction and relaxation.

All three elements are tightly interwoven, and good coordination and dynamic posture will result only if all of them are functioning normally.

This report describes an approach to dynamic posture through exercises based on primitive motion patterns. Three sequences of movements have been shown in detail. The first is based on the diagonal reciprocal creeping, corresponding to the walking pattern of the horse. The second is the diagonal reciprocal creeping pattern, corresponding to the human gait. The third sequence is based on the type of movement seen in the positive and negative stretch reflex.

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SCHOOLS APPROVED FOR TRAINING PHYSICAL THERAPISTS

By Council on Medical Education and Hospitals of the American Medical Association

Name and Location of School	Medical Director	Technical Director	Entrance Requirements	Length in Months	Classes Start	Tuition	Certificate, Degree, Diploma
CALIFORNIA							
[1]Childrens Hospital Society, Los Angeles	Samuel S. Mathews	Sarah S. Rogers	a^-b^-d	14	Sept	$300	Cert. or Degree
[1]College of Medical Evangelists, Loma Linda, Calif.	F. R. Moor	R. Wm. Borden	a-b-c-d	12	Sept	$225	Cert. or Degree
[1]Univ. of So. California, Los Angeles	Charles L. Lawman	Charlotte W. Anderson	a-b-d-e-g^	14	Sept	$324	Cert. or Degree
[1]Univ. of California Med. School, San Francisco	Lucile M. Rising	Margery Wagner	d-e	15	Sept	$150[7]	Cert. or Degree
[1]Stanford Univ., Stanford (Palo Alto)	Wm. Northway	Lucille Daniels	a-d-e-f-g^	12	Jan June Sept	$120	Cert. or Degree
[1]Permanente Hospital, Oakland and Vallejo	O. L. Huddleston	Dorothy Haag	e-b-d	12	July	$300	Cert.
COLORADO							
[1]Univ. of Colorado Med. Center, Denver	Harold Dinken	Mary Lawrence	a-b-d	12	Sept	$300	Cert. or Degree
ILLINOIS							
Northwestern Univ. Med. School, Chicago	Emil D. W. Haeser	Gertrude Ruud	a-b-d-e	12	Oct	$450	Cert.
IOWA							
[1]Univ. of Iowa Med. School, Iowa City	W. D. Paul	Olive C. Furr	d^-e	12	Sept	$300	Cert.
KANSAS							
[1]Univ. of Kansas, School of Med., Kansas City	Donald L. Rose	Lilyan G. Warner & Ruth Mansfield	a-b-d-g^	12	Feb Sept	$90[7]	Cert. or Degree
MASSACHUSETTS							
Beaver-Boston School of Phys. Educ., Boston	Howard Moore	Constance K. Greene	d	36	Sept	$550	Degree
Simmons College, Boston	Wm. T. Green / Arthur L. Watkins	Janet B. Merrill	a^-b-d-g^	16-36	Sept	$650	Cert. or Degree
Boston Univ. College of Phys. Ed. for Women: Sargent College, Cambridge	Kenneth Christophe	Adelaide McGarrett	a-b-d-f-g^	12 4 yrs.	Sept	$405	Cert. or Degree
MINNESOTA							
[1]Univ. of Minnesota, Minneapolis	Frederic J. Kottke	Ruby M. Green	d-f-g	2 yrs.	Sept	$120[7]	Degree
[1]Mayo Clinic, Rochester	Earl C. Elkins	Harry Keown	a-b-c	12	Sept	None	Cert.
MISSOURI							
[1]Washington Univ. School of Medicine, Barnes Hospital, St. Louis	Sedgwick Mead	Beatrice F. Schulz	f	2 yrs.	Sept	$450[8]	Degree
[1]St. Louis Univ. School of Nursing, St. Louis	A. J. Kotkin	Sr. Mary Imelda	g^	36	Jan Sept	$300	Degree
NEW YORK							
Albany Hospital, Albany	John W. Ghormley	Catherine Graham	a-b-d	12	Sept	$350	Cert.
[1]Columbia Univ., New York	Wm. B. Snow	Fay Pinkerton	a-e-f 1 or 2 yrs.		Sept	$450	Cert. or Degree
[1]New York Univ. School of Education, New York	George G. Deaver	Elizabeth C. Addams	a^-b-d-g^	12	Sept	$540	Cert. or Degree
NORTH CAROLINA							
[1]Duke Univ. School of Med., Durham	Lenox Baker	Helen Kaiser	a^-b^-d	12	Oct	$300	Cert.
OHIO							
[1]Cleveland Clinic, Cleveland	S. G. Gamble	Mildred Heap	a-b-c	12	Oct	$300	Cert.
PENNSYLVANIA							
[1]D. T. Watson School of Physical Therapy, Leesdale	Jessie Wright	Kathryn Kelley	a^-b^-d	12	Oct	$300	Diploma
[1]Univ. of Pa. School of Auxiliary Med. Services, Div. of P.T., Philadelphia	George M. Piersol	Dorothy Bostikes	g^ / e	4 yrs. 12	Sept Sept	$900[8] $360[8]	Cert. & Degree Cert.
TEXAS							
[1]Hermann Hospital, School of Physical Therapy, Houston	Owen O. Selke, Jr.	Mary Elizabeth Kalb	a-b-d	12	Oct	$300	Cert. or Degree
[1]Univ. of Texas School of Med., Galveston	G. W. N. Eggers	Ruby Decker	a-b-d	12	Jan	$167[7]	Cert.
VIRGINIA							
[1]Baruch Center of Physical Med. of Medical College of Virginia, Richmond	Walter J. Lee	Susanne Hirt	a-b-c-g^	12	Sept	$375[3]	Cert. or Degree
WISCONSIN							
[1]Univ. of Wisconsin Med. School, Madison	Harry D. Beaman	Margaret Kuhli	a-b-f-g^	12	Sept	$120[9]	Cert. or Degree
MEDICAL DEPARTMENT—U. S. ARMY							
(a) Medical Field Service School, Brooke Army Medical Center, San Antonio, Texas, and	James E. Tate, Lt. Col., M.C.	Agnes P. Snyder, Major, WMSC	b-c	12[5]	Aug Mar	None	Cert.
(b) Fitzsimons General Hospital, Denver, Colorado, or	H. B. Lancombe, Colonel, M.C.	Olivia M. Cole, Captain, WMSC	Completion of Medical Field Service School				
(c) Walter Reed General Hospital, Army Medical Center, Washington, D. C.	John H. Kulberi, Lt. Col., M.C.	Brunetta Knobilton, Major, WMSC	Completion of Medical Field Service School				

Entrance Requirements

For One-year Course: a=Graduation from accredited school of nursing; b=Baccalaureate degree with major in physical education; c=Two years of college with science courses; d=Three years of college with science courses; e=Baccalaureate degree with required science courses.

For Two-year Course: f=Two years of college with science courses.

For Four-year Degree Course: g=High school graduation.

[1]Male students admitted.

[2]Nonresidents charged additional fee.

[3]Certificate granted by Childrens Hospital Society; degree granted by University of California at Los Angeles.

[4]Tuition reduced second semester of second year.

[5]3 months includes training at the Medical Field Service School and Fitzsimons or Walter Reed General Hospitals.

[6]Additional college science required.

[7]Plus additional University fees.

[8]Advanced standing students accepted.

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